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TRANSACTIONS

OF THE

COLLEGE OF PHYSICIANS

OF

PHILADELPHIA.

THIRD SERIES.

VOLUME THE TWENTY-SECOND.

PHILADELPHIA:
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1900.

NOTICE.

The present volume of TRANSACTIONS contains the papers read before the College from January, 1900, to December, 1900, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its TRANSACTIONS.

EDITED BY

WILLIAM ZENTMAYER.

DORNAN, PRINTER.

COLLEGE OF PHYSICIANS OF PHILADELPHIA.

1900.

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JOHN B. DEAVER, M.D.

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OF THE

PRESIDENTS OF THE COLLEGE FROM THE TIME OF ITS INSTITUTION.

ELECTED

1787 JOHN REDMAN
1805 WILLIAM SHIPPEN
1809 ADAM KUHN
1818 THOMAS PARKE
1835 THOMAS C. JAMES*
1835 THOMAS T. HEWSON
1848 GEORGE B. WOOD
1879 W. S. W. RUSCHENBERGER
1883 ALFRED STILLÉ
1884 SAMUEL LEWIS†
1884 J. M. DA COSTA
1886 S. WEIR MITCHELL
1889 D. HAYES AGNEW
1892 S. WEIR MITCHELL
1895 J. M. DA COSTA
1898 JOHN ASHHURST, JR.
1900 W. W. KEEN, M.D.

* Died four months after his election.
† Resigned on account of ill-health.

FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA.

DECEMBER, 1900.

* Non-resident Fellows.
† Fellows who have commuted dues.

ELECTED

*1883. Abbot, Griffith E., Ph.D., M.D., Washington, D. C.

1892. Abbott, Alex. C., M.D., Professor of Hygiene and Bacteriology, and Director of the Laboratory of Hygiene in the University of Pennsylvania.

1876. Alison, Robert H., M.D., Attending Physician to the Bryn Mawr Hospital.

1873. Allis, Oscar H., M.D., Surgeon to the Presbyterian Hospital.

1896. Allyn, Herman B., M.D., Instructor in Physical Diagnosis in the University of Pennsylvania; Physician to St. Joseph's and the Philadelphia Hospitals; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania.

1888. Anders, James M., M.D., LL.D., Professor of the Theory and Practice of Medicine and Clinical Medicine in the Medico-Chirurgical College; Physician to the Medico-Chirurgical and Samaritan Hospitals.

1869. Andrews, T. Hollingsworth, M.D., Medical Director of the Bureaus of Police and Fire, and Commandant of the Philadelphia Emergency Corps of the Department of Public Safety.

1896. Angney, William M., M.D., Physician to the House of Mercy (Home for Male Consumptives); Consulting Physician to the Hospital for Diseases of the Lungs at Chestnut Hill.

ELECTED

*1882. Ashbridge, Richard, M.D., West Whiteland, Pa.

1865. Ashhurst, Samuel, M.D., Surgeon to the Children's Hospital.

1893. Ashton, Thomas G., M.D., Physician to the Philadelphia Hospital; Assistant Physician to the Jefferson Medical College Hospital; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania.

1857. Atlee, Walter Franklin, A.M., M.D., Corresponding Member of La Société des Sciences Médicales de Lyons; Consulting Surgeon to St. Luke's Hospital, Bethlehem; Visiting Physician to the Preston Retreat.

1852. Bache, Thomas Hewson, M.D.

1883. Baer, Benjamin F., M.D., Professor of Gynecology in the Philadelphia Polyclinic.

†1892. Baker, George Fales, B.S., M.D.

1879. Baker, Washington H., M.D., Obstetrician to the Maternity Hospital.

1889. Baldy, John Montgomery, M.D., Professor of Gynecology in the Philadelphia Polyclinic; Surgeon to the Gynecean Hospital and to the Gynecological Out-patient Department of the Pennsylvania Hospital; Consulting Surgeon to the Frederick Douglass Memorial Hospital.

1898. Balliet, Tilghman M., A.M., M.D., Professor of Therapeutics at Dartmouth College, Hanover, N. H., Physician to the Old Man's Home.

1880. Bartholow, Roberts, M.D., Professor (Emeritus) of Materia Medica, General Therapeutics and Hygiene in the Jefferson Medical College.

1894. Barton, James M., M.D., Surgeon to the Jefferson Medical College Hospital and to the Philadelphia Hospital.

1883. Baum, Charles, A.M., M.D., Ph.D.

1883. Beates, Henry, M.D.

1860. Benner, Henry D., M.D.

1874. Bennett, W. H., M.D., Physician-in-Charge to the Seashore Home for Invalid Children, and to the Seaside House for Invalid Women, Atlantic City; formerly Physician to the Episcopal Hospital, and Physician-in-Charge to St. Christopher's Hospital for Children.

1896. Beyea, Henry D., M.D., Instructor in Gynecology and As-

ELECTED

sistant Demonstrator of Obstetrics in the University of Pennsylvania; Assistant Surgeon to the Gynecean Hospital.

†1884. Biddle, Alexander W., M.D.

1884. Biddle, Thomas, M.D.

*1866. Black, J. J., M.D., New Castle, Del.

1894. Bliss, Arthur Ames, M.D., Laryngologist and Aurist to the German Hospital; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb.

*1867. Boardman, Charles H., M.D., Evanston, Illinois.

1894. Bochroch, Max H., M.D., Instructor in Electro-therapeutics and Chief Clinical Assistant in the Nervous Department of the Jefferson Medical College Hospital; Neurologist to the Out-patient Department of St. Joseph's Hospital.

1896. Boger, John A., M.D., Surgeon to St. Mary's and the Samaritan Hospitals; Surgeon to the Dispensary of the Episcopal Hospital.

1891. Boyd, George M., M.D., Clinical Professor of Obstetrics in the Medico-Chirurgical College; Physician to the Philadelphia Lying-in Charity Hospital and Nurse School.

†1884. Bradford, T. Hewson, M.D.

1856. Brinton, John H., M.D., Professor of the Practice of Surgery and of Clinical Surgery in the Jefferson Medical College; Consulting Surgeon to St. Joseph's Hospital and to the Southwestern Hospital of Philadelphia.

1891. Brinton, Lewis, M.D., Visiting Physician to the Nervous Department of the Howard Hospital.

1900. Brinton, Ward, M.D., Demonstrator of Physical Diagnosis in the Jefferson Medical College; Physician to the Dispensary of St. Christopher's Hospital for Children; Assistant in the Medical Dispensary of the Jefferson Medical College Hospital.

1887. Brubaker, Albert P., M.D., Professor of Physiology in the Pennsylvania College of Dental Surgery; Adjunct Professor of Physiology and Hygiene in the Jefferson Medical College; Lecturer on Anatomy and Physiology in the Drexel Institute.

*1890. Brush, Edward N., M.D., Medical Superintendent of the Shepherd and Enoch Pratt Hospital, Towson, Md.

*1851. Bullock, William R., M.D., Wilmington, Del.

ELECTED

1870. Burnett, Charles H., M.D., Professor (Emeritus) of Otology in the Philadelphia Polyclinic; Clinical Professor of Otology in the Woman's Medical College; Aural Surgeon to the Presbyterian Hospital; Consulting Aurist to the Pennsylvania Institution for the Deaf and Dumb.

1892. Burr, Charles W., M.D., Neurologist to the Philadelphia Hospital.

1886. Cadwalader, Charles E., M.D.

1895. Carpenter, John T., M.D.

*1897. Carter, William S., M.D., Professor of Physiology in the University of Texas.

1892. Cattell, Henry W., A.M., M.D., Director of the Josephine M. Ayer Clinical Laboratory of the Pennsylvania Hospital; Editor of the *International Clinics*.

*1892. Cerna, David, M.D., Ph.D., Galveston, Texas, Demonstrator of Physiology in the Department of Medicine of the University of Texas; Corresponding Fellow of the Sociedad Española de Higiene of Madrid.

1900. Chance, Burton Kollock, M.D., Assistant Surgeon to the Wills Eye Hospital.

1885. Chapin, John B., M.D., Physician-in-Chief to the Pennsylvania Hospital for the Insane.

1880. Chapman, Henry C., M.D., Professor of the Institutes of Medicine and of Medical Jurisprudence in the Jefferson Medical College.

1900. Chase, Robert Howland, A.M., M.D., Superintendent of the Friends' Asylum for the Insane.

1868. Cheston, D. Murray, M.D.

1897. Cheston, Radcliffe, M.D.

1899. Clark, John G., M.D., Professor of Gynecology in the University of Pennsylvania; Gynecologist-in-Chief to the University Hospital.

1897. Claxton, Charles, A.M., M.D.

1872. Cleemann, Richard A., M.D.

1896. Cleveland, Arthur H., M.D., Clinical Professor of Laryngology in the Medico-Chirurgical College; Surgeon-in-Charge of the Ear, Nose, and Throat Dispensary of the Presbyterian Hospital.

ELECTED

*1842. Clymer, Meredith, M.D., New York.

1871. Cohen, J. Solis, M.D., Professor (Emeritus) of Diseases of the Throat and Chest in the Philadelphia Polyclinic; Professor (Honorary) of Laryngology in the Jefferson Medical College; Physician to the Home for Consumptives.

1888. Cohen, Solomon Solis, M.D., Professor of Medicine and Therapeutics in the Philadelphia Polyclinic; Lecturer on Clinical Medicine in the Jefferson Medical College; Physician to the Philadelphia, the Rush and the Polyclinic Hospitals; Consulting Physician to the Jewish Hospital.

1898. Coles, Stricker, M.D., Demonstrator of Obstetrics in the Jefferson Medical College; Assistant Obstetrician to the Jefferson Maternity.

1895. Cross, William A., M.D., Consulting Physician to the Jewish Hospital.

1884. Curtin, Roland G., M.D., Consulting Physician to the Rush, St. Timothy's and Douglass Hospitals.

1884. Da Costa, John C., M.D., Gynecologist to the Jefferson Medical College Hospital; Consulting Gynecologist to St. Agnes's Hospital; President of the Philadelphia Obstetrical Society.

1896. Da Costa, John Chalmers, M.D., Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Surgeon to the Philadelphia and St. Joseph's Hospitals.

1887. Daland, Judson, M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Assistant Physician to the Hospital of the University of Pennsylvania; Professor of Clinical Medicine in the Philadelphia Polyclinic; Consulting Physician to the Kensington Hospital for Women.

1859. Darrach, James, M.D., Consulting Surgeon to the Germantown Hospital.

1896. Davis, Charles N., M.D, Assistant Physician to the Dispensary for Diseases of the Skin, and Assistant Surgeon in the Dispensary for Genito-Urinary Diseases in the Hospital of the University of Pennsylvania; Physician to the Department for Skin Diseases of the Northern Dispensary.

1888. Davis, Edward P., A.M., M.D., Professor of Obstetrics in the Jefferson Medical College and in the Philadelphia Polyclinic; Visiting Obstetrician to the Jefferson and Polyclinic Hos-

ELECTED

pitals; Obstetrician and Gynecologist to the Philadelphia Hospital; Member of the American Gynecological Society, the American Pedriatic Society and of the International Congress of Obstetrics and Gynecology.

1889. Davis, Gwilym G., M.D. (Univ. of Penna. and Goettingen), M.R.C.S. Eng., Assistant Professor of Applied Anatomy in the University of Pennsylvania; Surgeon to the Episcopal, St. Joseph's and Orthopædic Hospitals.

1900. Davisson, Alex. Heron, M.D.

1894. Deaver, Harry C., M.D., Surgeon to the Episcopal, St. Mary's and Samaritan Hospitals and to St. Christopher's Hospital for Children.

1887. Deaver, John B., M.D., Surgeon-in-Chief to the German Hospital; Consulting Surgeon to the Germantown Hospital.

1892. Deaver, Richard Wilmot, M.D.

1885. Dercum, Francis X., M.D., Clinical Professor of Neurology in the Jefferson Medical College; Neurologist to the Philadelphia Hospital; Consulting Neurologist to St. Agnes's and the Jewish Hospitals, and to the State Asylum for the Chronic Insane of Pennsylvania.

1891. Dixon, Samuel G., M.D., President and Executive Curator of the Academy of Natural Sciences of Philadelphia; Member of the Council of the American Philosophical Society; Member of the Board of Trustees of the Wistar Institute of Anatomy; Member of the Board of Managers of the Philadelphia Zoölogical Society.

1891. Dixon, William C., M.D., Physician to the Industrial Home for Blind Women; Physician to the Shelter for Colored Orphans; Member of Consulting Staff of the Philadelphia Home for Incurables; Examiner of Insane Patients to the Philadelphia Hospital.

1896. Donnellan, P. S., M.D., L.R.C.S. and P., Ireland; Laryngologist to St. Agnes's Hospital; Medical Examiner of the Mutual Life Insurance Company of New York.

1897. Dorland, W. A. Newman, M.D., Instructor in Gynecology in the Philadelphia Polyclinic and College for Graduates in Medicine; Assistant Demonstrator of Obstetrics in the University of Pennsylvania.

1893. Downs, Norton, M.D.

ELECTED

1864. Downs, R. N., M.D.

1884. Drysdale, T. M., M.D.

1864. Duer, Edward L., A.M., M.D., Gynecologist to the Presbyterian Hospital; Consulting Obstetrician to the Maternity Hospital and to the Preston Retreat.

1897. Duer, S. Naudain, M.D., Physician to the Dispensary of the Presbyterian Hospital.

1871. Duhring, L. A., M.D., Professor of Skin Diseases in the University of Pennsylvania.

1881. Dulles, Charles Winslow, M.D., Lecturer on the History of Medicine in the University of Pennsylvania; Surgeon to the Rush Hospital.

1863. Dunglison, Richard J., M.D.

*1871. Dunglison, Thomas R., M.D., Rosny sous Bois (Seine), France.

1860. Dunton, William R., M.D., Consulting Physician to the Germantown Hospital.

1899. Edsall, David L., M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Assistant Physician to the University Hospital; Associate of the Pepper Laboratory of Clinical Medicine; Physician to the Home for Incurables and to St. Christopher's Hospital for Children; Pathologist to the Methodist Hospital.

*1887. Edwards, William A., M.D., Physician to the Coronado Hospital, Coronado, California.

1896. Ely, Thomas C., A.M., M.D.

1893. Eshner, Augustus A., M.D., Professor of Clinical Medicine in the Philadelphia Polyclinic and College for Graduates in Medicine; Physician to the Philadelphia Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases.

*1880. Eskridge, J. T., M.D., Neurologist to St. Luke's Hospital, and Consulting Alienist and Neurologist to the Arapahoe County Hospital, Denver, Colorado.

1868. Evans, Horace Y., M.D., Physician to the Charity Hospital.

1894. Faries, Randolph, M.D., Surgeon to the Orthopedic Dispensary of the Hospital of the University of Pennsylvania; Director of Physical Education in the Protestant Episcopal Academy.

ELECTED

1893. Farr, William W., M.D.

1884. Fenton, Thomas H., M.D., Medical Director and Senior Ophthalmologist of the Union Mission Hospital; Ophthalmologist to St. Vincent's Home, to the Home for Aged Couples, to the Baptist Home and to the House of the Good Shepherd.

1884. Fisher, Henry M., M.D., Physician to the Episcopal Hospital; Physician to the Out-patient Department of the Pennsylvania Hospital.

1900. Flexner, Simon, M.D., Professor of Pathology in the University of Pennsylvania; Pathologist to the Philadelphia Hospital; Member of the Association of American Physicians; Corresponding Member of La Societa Medico-Chirurgica di Bologna.

1888. Flick, Lawrence F., M.D.

1862. Forbes, William S., M.D., Professor of Anatomy in the Jefferson Medical College; Clinical Surgeon to the Jefferson Medical College Hospital.

†1885. Fox, Joseph M., M.D., Leesburg, Va.

1897. Frazier, Charles H., M.D., Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the University, Philadelphia and Howard Hospitals; Surgeon to the Home for Crippled Children.

†1890. Freeman, Walter J., M.D., Professor of Laryngology in the Philadelphia Polyclinic; Laryngologist to the Children's and Orthopædic Hospitals; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb.

1900. French, Morris S., M.D.

1893. Friebis, George, M.D., Ophthalmic Surgeon to the Lutheran Home and Orphanage, Mt. Airy.

1899. Furness, William H., 3d, M.D.

1889. Fussell, M. Howard, M.D., Chief Physician to the Medical Dispensary of the Hospital of the University of Pennsylvania; Instructor in Clinical Medicine in the University of Pennsylvania.

'1899. Gamble, Robert G., M.D., One of the Attending Physicians to the Bryn Mawr Hospital.

1873. Gerhard, George S., M.D.

ELECTED

1864. Getchell, F. H., M.D.

1892. Gibb, Joseph S., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Surgeon to the Ear, Nose and Throat Department of the Episcopal Hospital.

1899. Gibbon, John H., M.D., Surgeon to the Bryn Mawr Hospital; Assistant Surgeon to the Jefferson Medical College Hospital; Surgeon to the Out-patient Departments of the Pennsylvania and the Children's Hospitals.

1897. Girvin, John H., M.D., Physician for Diseases Peculiar to Women in the Presbyterian Hospital; Assistant Demonstrator of Obstetrics in the University of Pennsylvania.

1889. Githens, William H. H., M.D., Visiting Physician to the Sheltering Arms.

1894. Gleason, E. B., M.D., Clinical Professor of Otology in the Medico-Chirurgical College; Surgeon-in-Charge of the Nose, Throat and Ear Department of the Northern Dispensary.

*1893. Gobrecht, William H., M.D., Washington, D. C.

1884. Godey, Harry, M.D.

1893. Goodell, W. Constantine, M.D.

†1897. Gould, George M., A.M., M.D.

1894. Graham, Edwin E., M.D., Clinical Professor of Diseases of Children in the Jefferson Medical College; Physician to the Franklin Reformatory Home.

1885. Graham, John, M.D.

1891. Green, Walter D., A.M., M.D.

1883. Griffith, J. P. Crozer, M.D., Clinical Professor of the Diseases of Children in the University of Pennsylvania; Physician to St. Agnes's, the Children's and the Methodist Hospitals.

1871. Grove, John H., M.D., Consulting Surgeon to St. Agnes's Hospital.

*1889. Guitéras, John, M.D.

*1893. Hamill, Robert H., M.D., Summit, N. J.

1894. Hamill, Samuel McC., M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Physician to the

ELECTED

Medical Dispensary of the Hospital of the University of Pennsylvania; Physician to St. Christopher's Hospital for Children; Pediatrist to the Howard Hospital.

1897. Hand, Alfred, Jr., M.D., Physician to the Out-patient Department of the Methodist, Children's and Polyclinic Hospitals; Pathologist to the Children's Hospital.

1886. Hansell, Howard F., M.D., Clinical Professor of Ophthalmology in the Jefferson Medical College; Professor of Diseases of the Eye in the Philadelphia Polyclinic; Consulting Ophthalmologist to the Chester County Hospital and to the Frederick Douglass Memorial Hospital.

1889. Hare, Hobart A., M.D., Professor of Therapeutics in the Jefferson Medical College; Physician to the Jefferson Medical College Hospital.

1865. Harlan, George C., M.D., Surgeon to Wills Eye Hospital and to the Eye and Ear Department of the Pennsylvania Hospital; Professor (Emeritus) of Diseases of the Eye in the Philadelphia Polyclinic; Consulting Ophthalmologist to the Pennsylvania Institution for the Blind and to the Pennsylvania Institution for the Deaf and Dumb.

1885. Harte, Richard H., M.D., Surgeon to the Pennsylvania and Episcopal Hospitals; Consulting Surgeon to St. Mary's and St. Timothy's Hospitals.

1888. Hartzell, Milton B., M.D., Instructor in Dermatology in the University of Pennsylvania; Dermatologist to the Methodist Episcopal Hospital.

1872. Hays, I. Minis, M.D.

1882. Hearn, W. Joseph, M.D., Clinical Professor of Surgery in the Jefferson Medical College; Surgeon to the Philadelphia Hospital.

1884. Henry, Frederick P., M.D., Physician to the Philadelphia Hospital; Professor of the Principles and Practice of Medicine in the Woman's Medical College of Pennsylvania.

1891. Hewson, Addinell, A.M., M.D., Demonstrator of Anatomy in the Jefferson Medical College; Surgeon to the Dispensary of the Episcopal Hospital; Surgeon to St. Timothy's Hospital.

1872. Hinckle, A. G. B., M.D.

ELECTED

1897. Hinckle, William M., M.D., Lecturer on the Anatomy and Physiology of the Vocal Organs in the National School of Elocution and Oratory.

1892. Hinsdale, Guy, M.D., Consulting Physician to the Presbyterian Orphanage; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases and to the Presbyterian Hospital.

1888. Hirsh, A. Bern., M.D., Physician to the Home for Aged Couples.

1888. Hirst, Barton Cooke, M.D., Professor of Obstetrics in the University of Pennsylvania; Gynecologist to the Philadelphia and Howard Hospitals.

1894. Hoch, William R., M.D., Instructor in Laryngology in the University of Pennsylvania; Laryngologist to the Methodist Episcopal Hospital.

1885. Holland, James W., M.D., Professor of Medical Chemistry and Toxicology in the Jefferson Medical College.

†1879. Hopkins, William Barton, M.D., Surgeon to the Pennsylvania Hospital,

1888. Horwitz, Orville, M.D., Professor of Genito-urinary Diseases in the Jefferson Medical College; Surgeon to the Philadelphia Hospital and to the State Hospital for the Insane; Consulting Surgeon to the Hayes Mechanics' Home.

1868. Howell, Samuel B., M.D., Professor of Chemistry in the Medico-Chirurgical College.

1892. Hughes, William E., M.D., Professor of Clinical Medicine in the Medico-Chirurgical College; Visiting Physician to the Philadelphia and Medico-Chirurgical Hospitals; Pathologist to the Presbyterian Hospital.

1898. Hutchinson, James P., M.D., Surgeon to the Dispensaries of the Episcopal, Methodist and Children's Hopitals.

1871. Ingham, James V., M.D.

*1885. Jackson, Edward, M.D., Denver, Colorado, Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic.

1887. Jayne, Horace, M.D., Ph.D., Professor of Zoölogy in the University of Pennsylvania; Director of the Wistar Institute of Anatomy and Biology,

ELECTED

1898. Johnson, Russell H., M.D., Physician to the Pennsylvania Institution for the Deaf and Dumb.

1900. Jones, Charles James, A.M., M.D.

1899. Jopson, John H., M.D., Surgeon to the Dispensaries of the Episcopal, Presbyterian and Children's Hospitals; Visiting Physician to the Philadelphia Home for Incurables.

1885. Judd, Leonardo Da Vinci, M.D.

1900. Judson, Charles F., A.B., M.D., Physician to the Episcopal Hospital; Physician to the Out-patient Departments of the Children's, the German and St. Christopher's Hospitals.

1886. Jurist, Louis, M.D.

†1867. Keen, William W., M.D., LL.D., F.R.C.S. (Hon.), Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Surgeon to the Jefferson Medical College Hospital; Membre Correspondant Étranger de la Société de Chirurgie de Paris; Membre Honoraire de la Société Belge de Chirurgie.

1897. Kelly, Aloysius O. J., M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Assistant Physician to the University Hospital; Clinical Professor of Pathology in the Woman's Medical College of Pennsylvania; Visiting Physician to St. Mary's and St. Agnes's Hospitals; Pathologist to the German Hospital.

*1887. Kelly, Howard A., M.D., Professor of Gynecology in Johns Hopkins University and Gynecologist to the Johns Hopkins Hospital, Baltimore, Md.

1898. Kempton, Augustus F., M.D.

*1844. King, Charles R, M.D., Andalusia, Pa.

1895. Kneass, Samuel S., M.D., Associate in the William Pepper Laboratory of Clinical Medicine in the University of Pennsylvania.

1900. Krusen, Wilmer, M.D., Instructor in Gynecology in the Jefferson Medical College; Assistant Gynecologist and Chief of the Gynecological Dispensary of St. Joseph's Hospital; Consulting Gynecologist to the Children's Hospital in Germantown.

ELECTED

1897. Kyle, D. Braden, M.D., Clinical Professor of Laryngology, Rhinology and Otology in the Jefferson Medical College; Consulting Laryngologist, Rhinologist and Otologist to St. Agnes's Hospital and to the Philadelphia Home for Incurables; Laryngologist to the New Jersey Training School for Feeble-minded Children; Bacteriologist to the Orthopædic Hospital and Infirmary for Nervous Diseases.

*1892. Lainé, Damaso T., M.D., Havana, Cuba.

1865. La Roche, C. Percy, M.D.

1887. Leaman, Henry, M.D.

1893. Le Conte, Robert G., M.D., Surgeon to the Pennsylvania, the Children's and the Bryn Mawr Hospitals.

1883. Leffmann, Henry, A.M., M.D., D.D.S., Professor of Chemistry and Toxicology in the Woman's Medical College of Pennsylvania; Professor of Chemistry in the Wagner Free Institute of Science; Member of the Society of Public Analysts.

1892. Leidy, Joseph, M.D., Physician to the Out-patient Department of the Pennsylvania Hospital; Consulting Physician to the Pennsylvania Training School for Feeble-minded Children.

1855. Lewis, Francis W., M.D.

1877. Lewis, Morris J., M.D., Physician to the Children's Hospital, to the Orthopædic Hospital and Infirmary for Nervous Diseases and to the Pennsylvania Hospital.

1886. Lloyd, J. Hendrie, M.D., Neurologist to the Philadelphia Hospital; Physician to the Methodist Episcopal Hospital and to the Home for Crippled Children; Consulting Neurologist to the State Asylum for the Chronic Insane of Pennsylvania and to the Pennsylvania Training School for Feeble-minded Children.

1900. Lodge, John W., M.D., Consulting Physician to the Bryn Mawr Hospital.

1893. Longaker, Daniel, M.D.

1877. Longstreth, Morris, M.D.

1900. McCarthy, Daniel J., M.D., Instructor in Neurology in the Philadelphia Polyclinic; Associate of the William Pepper Laboratory of Clinical Medicine.

ELECTED

1875. McClellan, George, M.D., Consulting Surgeon to the Howard Hospital.

1895. McFarland, Joseph, M.D., Professor of Pathology and Bacteriology in the Medico-Chirurgical College; Pathologist to the Medico-Chirurgical and the Philadelphia Hospitals.

1900. McReynolds, Robert Phillips, M.D., One of the Resident Chiefs and one of the Chiefs of the Gynecological Dispensary of the Presbyterian Hospital.

1886. MacCoy, Alexander W., M.D., Surgeon for Diseases of the Nose and Throat in the Out-patient Department of the Pennsylvania Hospital; Member of the American Laryngological Society.

1896. Makuen, G. Hudson, M.D., Professor of Defects of Speech in the Philadelphia Polyclinic; Laryngologist to St. Mary's Hospital and to the Frederick Douglass Memorial Hospital; Visiting Consultant on Defects of Speech to the New Jersey Training School for Feeble-minded Children.

*1885. Mallet, John William, M.D., Ph.D. (Goett.), LL.D. (Princeton), F.R.S., Professor of Chemistry in the University of Virginia.

1898. Marshall, George Morley, M.D., Laryngologist to the Philadelphia Hospital; Physician and Laryngologist to St. Joseph's Hospital.

1893. Marshall, John, M.D., Professor of Chemistry and Toxicology in the University of Pennsylvania.

1889. Martin, Edward, M.D., Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the University, Howard, St. Agnes's, Philadelphia and Bryn Mawr Hospitals.

1885. Mays, Thomas J., M.D., Professor of Diseases of the Chest and of Experimental Therapeutics in the Philadelphia Polyclinic; Visiting Physician to the Rush Hospital.

*1868. Mears, J. Ewing, M.D.

1875. Meigs, Arthur V., M.D., Physician to the Pennsylvania Hospital; Consulting Physician to the Pennsylvania Institution for the Instruction of the Blind.

*1884. Mifflin, Houston, M.D., Columbia, Pa.

1894. Miller, D. J. Milton, M.D., Physician to the Episcopal Hospital; Assistant Physician to the Children's Hospital.

ELECTED

1881. Mills, Charles K., M.D., Professor of Mental Diseases and of Medical Jurisprudence in the University of Pennsylvania; Clinical Professor of Neurology in the Woman's Medical College of Pennsylvania; Neurologist to the Philadelphia Hospital.

†1888. Mitchell, John K., M.D., Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Assistant Neurologist to the Presbyterian Hospital; Attending Physician to the Pennsylvania Training School for Feeble-minded Children.

1856. Mitchell, S. Weir, M.D., M. Nat. Ac. Sci., LL.D. (Harvard, Edinburgh and Princeton); M.D. *Honoris Causa* (Bologna, Italy); Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Physician to the Presbyterian Hospital.

1882. Montgomery, Edward E., M.D., Professor of Gynecology in the Jefferson Medical College; Gynecologist to the Jefferson and St. Joseph's Hospitals.

1863. Morehouse, George Read, M.D., Ph.D (Princeton), Consulting Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases.

1886. Morris, Caspar, M.D.

1893. Morris, Elliston J., M.D., Physician to the Episcopal Hospital, the Sheltering Arms and the Midnight Mission.

1883. Morris, Henry, M.D., Visiting Physician to St. Joseph's Hospital.

1856. Morris, J. Cheston, M.D.

1897. Morton, Samuel W., M.D.

1861. Morton, Thomas G., M.D., Senior Surgeon and President of the Medical Staff of the Pennsylvania Hospital; Fellow of the American Surgical Association.

1891. Morton, Thomas S. K., M.D., Professor of Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine; Surgeon to the Out-patient Department of the Pennsylvania Hospital; Consulting Surgeon to the Woman's Hospital and the Philadelphia Dispensary.

1864. Moss, William, M.D.

1890. Müller, Auguste F., M.D., Attending Physician to the Germantown Hospital.

ELECTED

1882. Musser, John H., M.D., Professor of Clinical Medicine in the University of Pennsylvania; Physician to the University, the Philadelphia and the Presbyterian Hospitals.

1896. Myers, T. D., M.D.

1886. Neff, Joseph S., M.D.

1887. Neilson, Thomas Rundle, M.D., Surgeon to the Episcopal Hospital and to St. Christopher's Hospital for Children; Professor of Genito-Urinary Surgery in the Philadelphia Polyclinic.

1899. Nicholson, William R., Jr., M.D., Instructor in Gynecology and Assistant Demonstrator of Obstetrics in the University of Pennsylvania; Obstetrician to the Maternity Hospital; Assistant Surgeon to the Gynecean Hospital.

1889. Noble, Charles P., M.D., Surgeon in-Chief to the Kensington Hospital for Women; Surgeon-in-Charge of the Department for Women of the Northern Dispensary and of the Union Mission Hospital; Clinical Professor of Gynecology in the Woman's Medical College of Pennsylvania; Lecturer on Gynecology in the Philadelphia Polyclinic.

1893. Noble, William H., M.D.

1898. Nolan, Edward J., M.D., Recording Secretary and Librarian of the Academy of Natural Sciences of Philadelphia.

1869. Norris, Herbert, M.D.

1865. Norris, Isaac, M.D.

1892. Norris, Richard C., M.D., Lecturer on Clinical and Operative Obstetrics in the University of Pennsylvania; Obstetrician-in-Charge to the Preston Retreat; Visiting Obstetrician to the Philadelphia Hospital; Gynecologist to the Methodist Hospital and Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary and Hospital.

1866. Norris, William F., M.D., Professor of Ophthalmology and Clinical Professor of Diseases of the Eye in the University of Pennsylvania; Surgeon to Wills Eye Hospital.

1884. Oliver, Charles A., A.M., M.D., Surgeon to Wills Eye Hospital; Ophthalmic Surgeon to the Philadelphia and the Presbyterian Hospitals; Consulting Ophthalmic Surgeon to St. Timothy's Hospital.

ELECTED

1884. O'Neill, J. Wilks, M.D.

*1885. Osler, William, M.D., Professor of Medicine in Johns Hopkins University and Physician to the Johns Hopkins Hospital, Baltimore, Md.

1897. Packard, Francis R., M.D., Dean of the Philadelphia Polyclinic and College for Graduates in Medicine; Surgeon for Diseases of the Ear in the Out-patient Department of the Pennsylvania Hospital; Instructor in Laryngology in the University of Pennsylvania.

1890. Packard, Frederick A., M.D., Visiting Physician to the Pennsylvania and Children's Hospitals.

1858. Packard, John H., M.D., Late Surgeon to the Pennsylvania Hospital; Surgeon Emeritus to St. Joseph's Hospital.

1898. Page, Henry F., M.D., Assistant Physician to the German Hospital and Physician to the Medical Dispensary of the same; Instructor in Clinical Medicine in the Woman's Medical College of Pennsylvania; Physician to the Baptist Home.

1882. Parish, William H., M.D., Professor of Obstetrics in the Dartmouth Medical College; Professor of Anatomy in the Woman's Medical College of Pennsylvania; Consulting Obstetrician to the Lying-in Charity Hospital; Consulting Surgeon to the Kensington Hospital; Medical Director and Gynecologist to St. Agnes's Hospital.

1899. Parke, William E., M.D., Assistant Surgeon in the Department for Diseases of Women of the Northern Dispensary; Clinical Assistant and Surgeon to the Dispensary of the Kensington Hospital for Women.

1898. Pearce, F. Savary, M.D., Clinical Professor of Nervous Diseases in the Medico-Chirurgical College of Philadelphia; Secretary of the Section on Nervous and Mental Diseases of the American Medical Association; Member of the American Climatological Association; Member of the Philadelphia Neurological Society.

†1889. Penrose, Charles Bingham, M.D.

1854. Penrose, R. A. F., M.D., LL.D., Professor (Emeritus) of Obstetrics and Diseases of Women and Children in the University of Pennsylvania.

ELECTED

1884. Perkins, Francis M., M.D., Ophthalmic Surgeon to St. Agnes's Hospital.

1899. Phillips, John L., M.D.

1883. Piersol, George A., M.D., Professor of Anatomy in the University of Pennsylvania.

1872. Porter, William G., M.D., Surgeon to the Presbyterian Hospital; Consulting Physician to the Philadelphia Dispensary and to the Educational Home for Boys.

1896. Posey, Wm. Campbell, M.D., Assistant Surgeon to Wills Eye Hospital; Ophthalmologist to the Howard and Epileptic Hospitals and the Home for Incurables; Consulting Ophthalmologist to the State Hospital for the Insane at Norristown.

1885. Potter, Thomas C., M.D.

1899. Potts, Charles S., M.D., Instructor in Nervous Diseases in the University of Pennsylvania; Physician to the Dispensary for Nervous Diseases and Assistant Neurologist to the University Hospital; Consulting Physician to the Hospital for the Insane of Atlantic County, New Jersey; Consulting Neurologist to the State Penitentiary, Eastern District of Pennsylvania.

†1899. Price, Joseph, M.D., Obstetric Physician to the Philadelphia Dispensary.

1887. Randall, B. Alexander, M.D., Clinical Professor of Diseases of the Ear in the University of Pennsylvania; Professor of Diseases of the Ear in the Philadelphia Polyclinic; Ophthalmic and Aural Surgeon to the Children's and Methodist Hospitals; Otologist to the Rush Hospital.

1887. Reed, Charles H., M.D.

1885. Reichert, Edward T., M.D., Professor of Physiology in the University of Pennsylvania.

1897. Rhein, John H. W., M.D., Neurologist to St. Agnes's Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Bacteriologist to the Pennsylvania Training School for Feeble-minded Children.

1891. Rhoads, Edward G., M.D.

1898. Riesman, David, M.D., Professor of Clinical Medicine in the Philadelphia Polyclinic; Instructor in Clinical Medicine in

ELECTED

the University of Pennsylvania; Physician to the Philadelphia Hospital; Neurologist to the Northern Dispensary.

1895. Ring, G. Oram, M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmic and Aural Surgeon to the Samaritan Hospital.

1891. Risley, S. D., M.D., Attending Surgeon to the Wills Eye Hospital; Professor of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; Member of the Board of Managers of the Pennsylvania Training School for Feeble-minded Children; Alumnus Manager of the University Hospital.

†1878. Roberts, John B., M.D., Professor of Anatomy and Surgery in the Philadelphia Polyclinic; Surgeon to the Methodist Hospital.

1899. Roberts, Walter, M.D., Instructor in Otology in the Philadelphia Polyclinic; Physician to the Ear, Nose and Throat Department of the Dispensary of St. Christopher's Hospital for Children; Clinical Assistant for Diseases of the Nose and Throat in the Out-patient Department of the Pennsylvania Hospital.

*1888. Robins, Robert P., M.D.

1900. Rodman, William L., M.D., Professor of the Principles of Surgery and Clinical Surgery in the Medico-Chirurgical College; Professor of the Principles and Practice of Surgery in the Woman's Medical College of Pennsylvania.

1898. Ross, George G., M.D., Assistant Surgeon to the German Hospital and Surgeon to the Out-patient Department of the same.

1897. Sailer, Joseph, M.D., Associate in the Pepper Clinical Laboratory in the University of Pennsylvania; Pathologist to the Pennsylvania Training School for Feeble-minded Children.

1900. Sajous, Charles E. de M., M.D.

†1866. Schäffer, Charles, M.D.

1899. Schamberg, Jay F., M.D., Associate in Diseases of the Skin in the Philadelphia Polyclinic; Dermatologist to the Union Mission Hospital.

1887. de Schweinitz, George E., M.D., Professor of Ophthalmology in the Jefferson Medical College; Ophthalmic Surgeon

ELECTED

to the Philadelphia Hospital; Ophthalmologist to the Orthopædic Hospital and Infirmary for Nervous Diseases; Consulting Ophthalmologist to the Philadelphia Polyclinic and College for Graduates in Medicine, to the Bryn Mawr Hospital and to the Chester County Hospital.

1895. Scott, J. Alison, M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Visiting Physician to the Pennsylvania Hospital; Physician to the Church Home for Children.

1892. Seiss, Ralph W., M.D., Professor of Otology in the Philadelphia Polyclinic; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb.

1888. Seltzer, Charles M., M.D.

1875. Seyfert, Theodore H., M.D.

1884. Shaffner, Charles, M.D., Ophthalmic Surgeon to the Presbyterian Hospital.

1897. Sharpless, W. T., M.D., Physician to the Chester County Hospital, West Chester, Pa.

1876. Shippen, Edward, A.M. (Princeton), M.D., Medical Director U. S. Navy (retired).

1891. Shober, John B., M.D., Gynecologist to the Philadelphia and Howard Hospitals; Assistant Gynecologist to the Gynecean Hospital.

1890. Shoemaker, George Erety, A.M., M.D., Gynecologist to the Methodist and the Presbyterian Hospitals.

†1893. Shoemaker, Harvey, M.D., Visiting Physician to the Sheltering Arms and to the Southern Home for Destitute Children; Assistant Physician to the German Hospital; Physician to the Out-patient Departments of the German and Pennsylvania Hospitals.

†1896. Shoemaker, William T., M.D., Assistant Ophthalmologist to the German Hospital and Ophthalmic Surgeon to the Out-patient Department of the same; Ophthalmic Surgeon to the Out-patient Department of the Presbyterian Hospital; Ophthalmologist to the Southern Home for Destitute Children.

1900. Shumway, Edward Adams, B.S., M.D.

1880. Simes, J. Henry C., M.D., Emeritus Professor of Genito-Urinary and Venereal Diseases in the Philadelphia Polyclinic.

ELECTED

1872. Sinkler, Wharton, M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Neurologist to the State Asylum for the Chronic Insane of Pennsylvania.

1895. Slocum, Harris A., M.D., Professor of Gynecology in the Philadelphia Polyclinic; Gynecologist to St. Clement's Hospital for Epileptics.

*1863. Smith, A. K., M.D., U. S. A. (retired), New York.

1895. Spellissy, Joseph M., M.D., Visiting Surgeon to St. Mary's Hospital; Surgeon to the Out-patient Departments of the Pennsylvania, Methodist and St. Agnes's Hospitals; Assistant Surgeon to the Orthopedic Dispensary of the University Hospital.

1897. Spiller, William G., M.D., Professor of Diseases of the Nervous System in the Philadelphia Polyclinic and College for Graduates in Medicine; Associate in the William Pepper Clinical Laboratory of the University of Pennsylvania; Neurologist to the New Jersey Training School for Feeble-minded Children; Pathologist to the Pennsylvania Training School for Feeble-minded Children and to the Pennsylvania Epileptic Hospital and Colony Farm.

1894. Stahl, B. Franklin, Ph.G., B.S., M.D., Instructor in Physical Diagnosis, and Lecturer on Dietetics of the Sick in the University of Pennsylvania; Visiting Physician to St. Agnes's Hospital; Neurological Registrar to the Philadelphia Hospital.

1875. Starr, Louis, M.D.

1898. Steele, J. Dutton, M.D., Instructor in Medicine, and Students' Physician in the University of Pennsylvania; Bacteriologist to the Presbyterian Hospital; Physician to the Church Home for Children.

1892. Steinbach, Lewis W., M.D., Professor of Clinical and Operative Surgery in the Philadelphia Polyclinic; Visiting Surgeon to the Philadelphia and the Jewish Hospitals.

1884. Stelwagon, Henry W., M.D., Ph.D., Clinical Professor of Dermatology in the Jefferson Medical College and in the Woman's Medical College; Physician to the Department for Skin Diseases of the Howard Hospital; Socio Corrispondente di la Societa Italiano di Dermatologia e Sifilografia.

ELECTED

1895. Stengel, Alfred, M.D., Professor of Clinical Medicine in the University of Pennsylvania; Physician to the Pennsylvania, Philadelphia and Children's Hospitals.

1888. Stewart, David D., M.D., Professor in the Philadelphia Polyclinic; Attending Physician to the Episcopal Hospital; Consulting Physician to the Kensington Hospital for Women.

1898. Stiles, George M., M.D.

1898. Stout, George C., M.D., Laryngologist and Aurist to St. Mary's Hospital and to the Children's Aid Society; Instructor in Otology in the Philadelphia Polyclinic and College for Graduates in Medicine.

1884. Stryker, Samuel S., M.D., Physician to the Presbyterian Hospital.

1900. Swan, John M., M.D., Demonstrator of Osteology and Assistant Demonstrator of Anatomy in the University of Pennsylvania; Dispensary Physician to the Presbyterian Hospital.

1898. Sweet, William M., M.D., Associate in Ophthalmology in the Philadelphia Polyclinic; Instructor in Ophthalmology, and Chief Clinical Assistant in the Out-patient Eye Department of the Jefferson Medical College; Ophthalmic Surgeon to the Phœnixville Hospital.

1900. Talley, James Ely, A.B., M.D., Physician to the Out-patient Department of the Presbyterian Hospital; Assistant and Consulting Physician to the Country Branch of the Children's Hospital.

1886. Taylor, John Madison, M.D., Neurologist to the Howard Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Assistant Physician to the Children's Hospital; Professor of Children's Diseases in the Philadelphia Polyclinic.

1887. Taylor, William J., M.D., Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases, and to St. Agnes's Hospital; Consulting Surgeon to the West Philadelphia Hospital for Women.

1886. Taylor, William L., M.D.

1867. Thomas, Charles Hermon, M.D.

1897. Thomson, A. G., M.D., Ophthalmic Surgeon to the Children's Hospital; Assistant Ophthalmic Surgeon to Wills Eye Hos-

ELECTED

pital; Assistant Ophthalmologist to the Orthopædic Hospital and Infirmary for Nervous Diseases.

†1869. Thomson, William, M.D., Emeritus Professor of Ophthalmology in the Jefferson Medical College; Surgeon to Wills Eye Hospital.

1896. Thorington, James, A.M., M.D., Professor of Diseases of the Eye in the Philadelphia Polyclinic.

1898. Thornton, Edward Q., M.D., Demonstrator of Therapeutics in the Jefferson Medical College.

1896. Toulmin, Harry, M.D., Assistant Medical Director of the Penn Mutual Life Insurance Company.

†1894. Tunis, Joseph Price, M.D., Formerly Assistant Demonstrator of Anatomy and of Surgery in the University of Pennsylvania; Surgeon to the Methodist Hospital.

1866. Tyson, James, A.M., M.D., Professor of Medicine in the University of Pennsylvania and Physician to the Hospital of the University of Pennsylvania; Physician to the Philadelphia Hospital; Member of the Association of American Physicians.

1897. Tyson, T. Mellor, M.D., Assistant Physician to the Hospital of the University of Pennsylvania; Physician to the Rush Hospital, the Philadelphia Lying-in Charity Hospital and the Children's Aid Society of Philadelphia.

1864. Vandyke, Edward B., A.M., M.D.

1873. Van Harlingen, Arthur, Ph.B. (Yale), M.D., Emeritus Professor of Dermatology in the Philadelphia Polyclinic; Dermatologist to the Children's Hospital.

1893. Vansant, Eugene Larue, M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Visiting Physician to the Throat, Nose and Ear Department of the Howard Hospital.

1897. Veasey, Clarence A., M.D., Adjunct Professor of Diseases of the Eye in the Philadelphia Polyclinic and College for Graduates in Medicine; Demonstrator of Ophthalmology in the Jefferson Medical College; Chief Clinical Assistant to the Ophthalmological Department of the Jefferson Medical College Hospital; Consulting Ophthalmologist to the Philadelphia Lying-in Charity Hospital.

†1883. Vinton, Charles Harrod, M.D.

ELECTED

1885. Walker, James B., M.D., Ph.D., Consulting Physician to the West Philadelphia Hospital for Women and Children.

1893. Warren, Joseph W., M.D., Associate Professor of Physiology in Bryn Mawr College.

1895. Watson, Arthur W., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Laryngologist to the Howard Hospital and to the Hospital for Diseases of the Lungs, Chestnut Hill.

1886. Watson, Edward W., M.D.

1875. Webb, William H., M.D.

1883. Welch, William M., M.D., Physician-in-Charge of the Municipal Hospital for Contagious Diseases; Consulting Physician to the Northern Dispensary and to the Northern Home for Friendless Children.

1897. Wells, William H., M.D., Adjunct Professor of Obstetrics and Diseases of Infancy in the Philadelphia Polyclinic; Demonstrator of Clinical Obstetrics in the Jefferson Medical College.

1893. Westcott, Thompson S., M.D., Instructor in Diseases in Children in the University of Pennsylvania; Visiting Physician to the Methodist Episcopal Hospital; Assistant Physician to the Children's Hospital.

1884. Wharton, Henry R., M.D., Clinical Professor of Surgery in the Woman's Medical College of Pennsylvania; Surgeon to the Presbyterian and Children's Hospitals; Consulting Surgeon to the Bryn Mawr Hospital, St. Christopher's Hospital for Children and the Pennsylvania Institution for the Deaf and Dumb.

1878. White, J. William, M.D., John Rhea Barton Professor of Surgery and Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the University Hospital.

1898. Whiting, Albert D., M.D., Assistant Surgeon and Registrar to the German Hospital, and Surgeon to the Out-patient Department of the same; Physician to the Southern Home for Destitute Children.

†1880. Willard, De Forest, M.D., Clinical Professor of Orthopedic Surgery in the University of Pennsylvania; Surgeon to the Presbyterian Hospital; Consulting Surgeon to the White and

ELECTED

the Colored Cripples' Homes, the Home for Incurables and the Atlantic City Hospital.

*1878. Williamson, Jesse, M.D., Wilmington, Delaware, one of the Surgeons to the Delaware Hospital.

1881. Wilson, H. Augustus, M.D., Emeritus Professor of Orthopedic Surgery in the Philadelphia Polyclinic; Clinical Professor of Orthopedic Surgery in the Jefferson Medical College; Consulting Orthopedic Surgeon to the Philadelphia Lying-in Charity Hospital and to the Kensington Hospital for Women.

1874. Wilson, James C., M.D., Professor of the Practice of Medicine and of Clinical Medicine in the Jefferson Medical College, and Physician to the Hospital of the same (Faculty Staff); Physician-in-Chief to the German Hospital; Attending Physician to the Pennsylvania Hospital.

1897. Wilson, W. Reynolds, M.D., Visiting Physician to the Philadelphia Lying-in Charity Hospital.

†1884. Wirgman, Charles, M.D., Physician to the Jefferson Medical College Hospital and to the Howard Hospital; Physician to the Out-patient Department of the Children's Hospital.

1893. Wolff, Lawrence, M.D., formerly Demonstrator of Chemistry in the Jefferson Medical College, Visiting Physician to the German Hospital and Clinical Professor of Medicine in the Woman's Medical College.

1893. Wood, Alfred C., M.D., Demonstrator of Surgery in the University of Pennsylvania; Assistant Surgeon to the University Hospital; Surgeon to the Philadelphia Hospital.

1900. Wood, George B., M.D., Instructor in Laryngology in the University of Pennsylvania; Assistant in the Clinic for Diseases of the Nose and Throat at the Polyclinic Hospital.

1865. Wood, Horatio C., M.D., LL.D. (Yale and Lafayette); Professor of Materia Medica and Therapeutics, and Clinical Professor of Diseases of the Nervous System in the University of Pennsylvania; Associate Fellow in Medicine and Surgery of the American Academy of Arts and Sciences; Member of the National Academy of Science.

1880. Woodbury, Frank, M.D., Associate in Laryngology in the Philadelphia Polyclinic.

1866. Woods, D. F., M.D., Physician to the Presbyterian Hospital.

ELECTED

1888. Woodward, Charles E., M.D., Secretary of the West Chester Board of Health; U. S. Examining Surgeon; Member of the Medical Staff of the Chester County Hospital.

†1897. Woodward, George, M.D.

1860. Wurts, Charles Stewart, M.D.

1868. Yarrow, Thomas J., M.D.

1889. Young, James K., M.D., Professor of Orthopedic Surgery in the Philadelphia Polyclinic and in the Woman's Medical College of Pennsylvania; Instructor in Orthopedic Surgery in the University of Pennsylvania and Assistant Orthopedic Surgeon to the University Hospital; Consulting Orthopedic Surgeon to the Hospital of the Good Shepherd, Radnor, Pa.

1894. Zentmayer, William, M.D., Assistant Surgeon to Wills Eye Hospital; Ophthalmologist to St. Mary's Hospital and to the House of Refuge.

1899. Ziegler, S. Lewis, M.D., Ophthalmic Surgeon to St. Joseph's Hospital.

1887. Ziegler, Walter M. L., A.M., M.D.

1895. Zimmerman, Mason W., M.D., Ophthalmic Surgeon to the Germantown Hospital and to St. Christopher's Hospital for Children.

ASSOCIATE FELLOWS.

[Limited to Fifty, of whom Twenty may be Foreigners.]

American.

ELECTED

1876. Billings, John S., M.D., U. S. A. (retired), New York.
1886. Bowditch, Henry P., M.D., Boston, Massachusetts.
1877. Chaillé, Stanford E., M.D., New Orleans, Louisiana.
1886. Cheever, David W., M.D., Boston, Massachusetts.
1896. Conner, Phineas Sanborn, M.D., Cincinnati, Ohio.
1893. Councilman, William T., M.D., Boston, Massachusetts.
1876. Davis, N. S., M.D., Chicago, Illinois.
1886. Draper, William H., M.D., New York.
1892. Emmet, Thomas Addis, M.D., New York.
1892. Fitz, Reginald H., M.D., Boston, Massachusetts.
1895. Fletcher, Robert, M.D., Washington, D. C.
1891. Jacobi, A., M.D., New York
1893 Kerr, John G., M.D., Canton, China.
1895. McBurney, Charles, M.D., New York.
1876. Moore, E. M., M.D., Rochester, New York.
1886. Reeve, John C., M.D., LL.D., Dayton, Ohio.
1886. Senn, Nicholas, M.D., Chicago, Illinois.
1896. Sternberg, George M., M.D., U. S. A., Washington, D. C.
1886. Thomas, T. Gaillard, M.D., New York.
1896. Tiffany, L. McLane, M.D., Baltimore, Maryland.
1894. Warren, J. Collins, M.D., Boston, Massachusetts.
1894. Weir, Robert F., M.D., New York.
1892. Welch, William H., M.D., Baltimore, Maryland.

Foreign.

1873. Acland, Henry W., M.D., F.R.S., Oxford, England.
1890. Baccelli, Guido, Rome, Italy.

ELECTED

1877. Barnes, Robert, M.D., London, England.
1894. Brunton, Sir T. Lauder, M.D., London, England.
1883. Fayrer, Sir Joseph, M.D., LL.D., F.R.S., London, England.
1899. Fraser, Thomas R., M.D., LL.D., F.R.C.P., F.R.S., Edinburgh, Scotland.
1883. Heath, Christopher, F.R.C.S., London, England.
1896. Jaccoud, Prof. S., M.D., Paris, France.
1874. Jackson, J. Hughlings, M.D., London, England.
1893. v. Jaksch, Rudolf, M.D., Prague, Bohemia.
1896. Leyden, Ernst, M.D., Berlin, Germany.
1877. Lord Lister, M.D., LL.D., F.R.S., London, England.
1873. Ogle, John W., M.D., London, England.
1898. Roddick, Thomas G., M.D., Montreal, Canada.
1896. Pye-Smith, P. H., M.D., London, England.
1869. Valcourt, Th. de, M.D., Cannes, France.
1892. Virchow, Rudolf, M.D., Berlin, Germany.

CORRESPONDING MEMBERS.

ELECTED

1880. Carrow, Flemming, M.D., United States.
1880. Chiara, Domenico, M.D., Florence, Italy.
1886. Dey, Kanny Lall, M.D., Calcutta, India.
1885. Rendu, Jean, M.D., Lyons, France.

NECROLOGICAL LIST.

FELLOWS.

WILLIAM A. HAMMOND (Non-resident),	January 6, 1900
ROBERT M. GIRVIN,	March 17, 1900
EDWARD O. SHAKESPEARE,	June 1, 1900
JOHN ASHHURST, Jr.,	July 7, 1900
THOMAS S. KIRKBRIDE, Jr.,	July 29, 1900
JOHN H. W CHESTNUT,	August 5, 1900
J. M. Da COSTA,	September 11, 1900
J. WILLOUGHBY PHILLIPS,	September 20, 1900
ALFRED STILLÉ,	September 24, 1900
ROSS R. BUNTING,	October 9, 1900
GEORGE A. MUEHLECK,	Oetober 25, 1900
MATTHEW J. GRIER,	October 27, 1900
EDWARD A. SMITH (Non-resident),	December 10, 1900

ASSOCIATE FELLOWS.

SIR T. GRAINGER STEWART,	February 3, 1900
JAMES T. WHITTAKER,	June 5, 1900
HUNTER McGUIRE,	September 19, 1900

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MEMOIR OF ALBERT FRICKÉ, M.D.

By THOMAS G. MORTON, M.D.

[Read February 7, 1900.]

It is seldom that the events of ordinary human life are so striking or conspicuous as to afford much material for the biographer or historian. In all walks of life we know that there are many who merit distinction compared with the few who achieve it. This is pre-eminently true of the medical profession, in whose ranks are many who have no ambition for a professional chair or for position as author or teacher, but who lead simple, plodding, seemingly uneventful lives, abundantly satisfied in their daily routine of devotion to duty, going about doing good, and relieving suffering and distress. All their lives they devote to the honorable service of the community in which their lot is cast, perhaps but little known except among their own particular surrounding; but when they are called away their memories are held in grateful remembrance by their brethren and by the many to whom they have so long and faithfully ministered.

Such a one was Albert Frické. Coming to Philadelphia nearly three score years ago, a stranger to our land and language, he by his industry, intelligence, and constant devotion to the duties of his profession became a prominent practitioner and a useful and honored citizen.

To those who had the privilege of his intimate acquaintance he was a valued friend. He lived more than half a century among us, devoted to the practice of the profession which he loved and to whose best interests he was ever loyal, and it is

eminently proper that at this time he should be the subject of our consideration.

Albert Fricke was born in Braunschweig, the capital of the Duchy of Brunswick, Northern Germany, on September 13, 1815; his father, George Fricke, Doctor of Laws, was Hofrath, or High Chancellor to the Court of Charles Frederick Augustus William, Duke of Brunswick. In 1814 he went to represent his august master at the great "Congress of Vienna," which met to regulate the affairs of Europe after the overthrow of Napoleon. When the Duke of Brunswick was deposed, in 1830, it necessitated the retirement of Dr. Fricke's father from the important duties of his position as Court Attorney, but he was so highly and universally esteemed that he was pensioned to the full extent of his salary of office until his death in 1848.

Dr. Fricke's mother, the born Baroness Julia Von Busse and Countess Königsberg, was a lady of gentle birth and was highly cultured.

Dr. Fricke's early education was received at the "Gymnasium," the preparatory school of the University at Göttingen. He then continued his studies at Hanover, Prussia; later he entered the University at Berlin, from which he graduated in medicine in 1839. He then entered the Prussian military service, which was required by law, and served the full term of two years. He settled in Berlin in 1841, intending if he could obtain his father's consent, which was obligatory, to practice his profession in that city. To this formal application he received a portentous document, duly signed by his father, attested by witnesses, also the signatures of State officials and seals of office, giving fatherly permission to the son to establish himself in the "Kingly Prussian State." At that time Dr. Fricke was offered a medical commission in the Russian army, but, finding that acceptance involved a service of ten years, he declined the appointment. After a residence of a year in Berlin he concluded to emigrate to America. His "passport" declared that he had completed his military service, and that he had fulfilled all duties to the State, and

that he was at liberty to leave the country. He sailed in a merchantman, and after a voyage of six weeks he safely landed at the port of Philadelphia in October, 1843. As one of the compensations for a slow voyage, it may be stated that during this time he never missed a day in having a swim in the ocean.

Dr. Frické took rooms on the south side of Cherry street, adjoining the Lutheran Church, on the corner of Fourth street. With Dr. Frické came his kinsman, Frederick Langenheim, well known as one of the first professional daguerreotypists (and subsequently photographer) in this country. Dr. Frické soon after his arrival removed to North Sixth street above Race, now No. 235, opposite Franklin Square, which, from the beautiful view of grass, trees, and fountain, with its forty jets, illuminated at night with gas, and more recently with electric light, was a source of continued pleasure to Dr. Frické, who resided here continuously for more than half a century. Although often urged to change to a more central location, here he always remained contented and happy. Soon after coming to Philadelphia, Dr. Frické became an American citizen, and he faithfully performed his duty as a voter until prevented by disability during his final illness. Dr. Frické had been for more than a year somewhat of an invalid, but he was able to be out in his carriage, and attended to office duties until within a week of his death, which occurred November 17, 1899, in the eighty-fifth year of his age. In 1848, Dr. Frické married Miss Lydia McKinley, by whom he had three children, one son and two daughters, all of whom died in early life. In 1853, Mrs. Frické died, and the following year he was again married. For his second wife he took a cousin of the first wife, Hester, widow of William A. Davis. The marriage took place on June 24, 1854. She died after a short illness on the 24th of December, 1893. There was no issue from this marriage.

Mr. Henry Cramer, a life-long friend of Dr. Frické, in a private letter, writes: "Dr. Frické is believed to have had a rudimentary acquaintance with English when he arrived here,

which he rapidly improved by his close contact with his English-speaking wife and her relatives. He was always ready to extend a helping hand for benevolent objects; unostentatious benevolence was a prominent feature of his character, often hidden under a rough surface. He responded with the same readiness to the call of the rich and the poor, be it day or night. In needy cases he was not slow to add, to his professional services, material help or assistance. Perhaps there are a few only who knew Dr. Frické as well as I did during the fifty years of our close friendship, and I do not hesitate to say he was 'a rough diamond.'"

With a more extended knowledge of English, Dr. Frické came more prominently before the medical men of the times, and naturally more known by the public generally.

Dr. Frické was well versed in the science and art of medicine, but he relied principally on his German medical education and subsequent German medical literature as authorities in his professional work, with which he was always in intimate touch.

In science he was more than a beginner; in German, French, and English literature he was well read; Goethe, Schiller, and Heine were his favorite authors. He was more than a fair microscopist, and, when it is said that he was an intimate personal friend of Leidy, it goes without saying that Frické was a man of no ordinary attainments.

Referring to their mutual interest in the natural sciences, Leidy, writing most affectionately to Dr. Frické from Brighton, England, in July, 1889, ends his letter saying: "I have been discontented ever since leaving home, having lost all interest in sight-seeing; beside examining museums in London, I spent some time in hunting up dealers in natural history specimens, and, among other things, I purchased a series of three mounted skeletons of the *gorilla*, *orang-outang*, and *chimpanzee*, which with a human skeleton I think will make an attractive row for our museum."

Dr. Frické was domestic in his habits, and he did not favor membership in clubs, seldom leaving home of an evening

unless to respond to professional calls or to attend, by rare exeeption, a meeting of a medical society. At the Academy of the Natural Sciences he was oeeasionally present, and was a member of the mieroseopical and biological seetion. He was a generous host, and delightfully entertained at his home on oecasions a few chosen friends, more especially each year on "Sylvester Eve." It would not be *possible* to speak of Fricke without a referenee to the coming together of friends on these oeeasions, which had been duly celebrated at least for more than thirty years.

This entertainment, being eondueted in genuine German style, ean hardly be deseribed, never forgotten. Upon the table there was more than abundance; a profusion and extravagance prevailed. The viands were prepared by German *chefs*, the table literally erowded with food most attractively displayed—salmon, venison, and other meats, pheasants, canvasbacks, quail, boned turkey, the birds arranged in pyramids, ornamented in various ways and eolors, German delicaeies of all sorts, curious German cakes, Rhine wines of rare vintage, choice champagnes, hot-house fruits, and iees, and the best Havanas.

The "punch," brewed from a recipe handed down from one generation to another in Dr. Frieké's family, eame in shortly before midnight; placed before the host was a beautiful old Bohemian glass service, a heritage brought from the Fatherland. The capacious bowl was filled with the steaming, odorous liquid from an old silver tankard, and the glasses were duly filled. When the "euckoo" eloek announeed the near approaeh of midnight the company rose, and, all standing around the table, welcomed the New Year amid German and English salutations.

On one oeeasion, on the sixty-fourth anniversary of Frieké's birth, a few of his friends were present, ineluding Joseph Leidy, Oswald Seidensticker, William Hunt, and the speaker, and, after a particulary enjoyable evening, Leidy, after a few welltimed remarks, decorated Frieké with a beautifully engraved gold medal in honor of the oeeasion On the obverse of the

medal—"A token of friendship and love," on the reverse, "September 13, 1879."

Dr. Frické was somewhat of a musician and had a fair voice, and soon after coming to the city became a member of the "Mænnerchor" Musical Society, organized in 1835, and with which he was associated more than fifty years. He also joined the German Society of Pennsylvania, established in 1764 for relieving and protecting German emigrants, which has for nearly a century and a half "faithfully fulfilled the object of its institution." In 1849, Dr. Frické became a member of the Philadelphia County Medical Society; ten years later he joined the Academy of the Natural Sciences. In 1864 he was elected a Fellow of the College of Physicians, and, at the time of his death, there was but one Fellow of this College his senior in age.

From the founding of the German Hospital in 1866 to 1874 he was a member of the medical staff and the senior physician. In 1872 he became a member of the American Medical Association. In 1880 he was elected a member of the Historical Society of Pennsylvania. On the 28th of August, 1862, Andrew G. Curtin, Governor of Pennsylvania, under instructions from the War Department at Washington, appointed Dr. Frické Surgeon for the County of Philadelphia—to act with the United States Commissioner to superintend drafting—in the examination of claims of citizens demanding exemption from military duty. He also served as an acting assistant surgeon in one of the United States army hospitals in Philadelphia during the Civil War.

Dr. Frické often referred to his very lonely life and surroundings, and once remarked that, so far as he knew, he had not a single living relative in the world.

Dr. Frické never appeared in haste or hurry; he was punctual at appointments, scrupulously neat in appearance, was always well dressed; his voice was clear, but he spoke with a slight German accent. His peculiarities of manner, address, and action increased with his years, and he was not apt in making new friends. He was about average height and of

good physique, and broad shoulders. His face was oval, with large German type of features, forehead high and open, florid complexion. In later years he increased much in weight. He was quite near-sighted, and generally wore gold spectacles, unless when reading or other near work, which he accomplished without such assistance, even to the close of his life.

His eyes were blue, hair and whiskers sandy; in later years he became quite bald, and wore a wig; his handwriting was exceptionally beautiful, large, neat, and distinct, each letter being carefully formed.

By close attention to his practice, care in making investments and avoidance of speculating and unnecessary display, Dr. Frické accumulated a moderate fortune.

By his will he directed that his house should be closed upon his death, but that no crape or other signs of mourning should be displayed upon it; that his body should be cremated, and that after such disposition, not before, a notice of his death should be published. After providing suitably for those who had a claim upon his bounty, he bequeathed one-half of his residuary estate, about $7000, to the Mutual Aid Association of the Philadelphia County Medical Society, of which he was one of the founders and one of its Vice-Presidents from its organization in 1878 until 1881, when he was elected a Director. In 1889, he was elected Chairman of the Executive Committee, which office he retained until April, 1897, when, on account of his age and impaired health, he felt obliged to resign.

At a recent meeting of the Directors the following minute was adopted:

The Association and this Board have lost in the death of Dr. Albert Frické one of the most earnest and lovable members of the Mutual Aid Association, a man who gave much thought and time to the business of the Association and who was intensely interested in its benevolent work. As an original member, Vice-President, and Director, Dr. Frické was always thoroughly imbued with the importance of the Association's work, and showed the liveliest interest in its progress. The provision which he has

made in his will for the relief fund of the Association is only another instance of his affection for his profession and his wise effort to aid in establishing a large benevolent fund for the relief of physicians and their widows and orphans. And it was

Resolved, That the Board enter upon its records its thorough appreciation of the valuable services which he has rendered the Association through these twenty years.

The impressively simple services over all that remained mortal of Albert Fricke ended by six of his friends, members of the Old Mænnerchor, singing in German:

"STILL RÜHT DEIN HERZ!"

"Silent thy heart, thy journey ending,
The pilgrim's staff thy hand let fall,
At Home thou hast found peace unending,
All care lies 'neath the grave's soft pall.

"Silent thy heart, so calmly resting,
The Father's house its shelter now,
Blest be thy slumbering, we trusting
Would whisper low, 'Adieu, adieu!'"

ANNUAL ADDRESS OF THE PRESIDENT.

By W. W. KEEN, M.D.

[Delivered December 5, 1900.]

I REGRET to call your attention to the fact, already stated in the Secretary's report, that our membership has increased in the past year by only two. One reason for this is the extraordinary mortality of the year.

May I ask you to rise while I read the necrology of the year?

NECROLOGY OF 1900.

FELLOWS.

Date of death.	Name.	Date of election to Fellowship.
Dec. 12, 1899	Dr. Emil Fischer	Elected a Fellow, October, 1866
Jan. 6, 1900	Dr. William A. Hammond (N. R.)	" " July, 1859
March 17, "	Dr. Robert M. Girvin	" " March, 1885
June 1, "	Dr. Edward O. Shakespeare	" " April, 1877
July 7, "	Dr. John Ashhurst, Jr.	" " July, 1863
July 29, "	Dr. Thomas S. Kirkbride, Jr.	" " January, 1900
Aug. 5, "	Dr. John H. W. Chestnut	" " February, 1897
Sept. 11, "	Dr. J. M. Da Costa	" " October, 1858
Sept. 20, "	Dr. J. Willoughby Phillips	" " April 1890
Sept. 21, "	Dr. Alfred Stillé	" " December, 1842
Oct. 9, "	Dr. Ross R. Bunting	" " June, 1887
Oct. 25, "	Dr. George A. Muehleck.	" " February, 1898
Oct. 27, "	Dr. Matthew J. Grier	" " October, 1870

ASSOCIATE FELLOWS.

Died.	Name.	Date of election.
	Foreign.	
Dec. 30, 1899	Sir James Paget	Elected an Associate Fellow in 1874
Feb. 3, 1900	Sir T. Grainger Stewart	" " " 1896
	American.	
June 5, 1900	Dr. James T. Whittaker, Cincinnati	" " " 1886
Sept 19, 1900	Dr. Hunter McGuire, Richmond	" " " 1886

Among these deceased Fellows are three ex-Presidents (Ashhurst, Da Costa, and Stillé), two ex-Presidents of the American Medical Association (Stillé and McGuire), and one former Surgeon-General of the Army (Hammond). The lives of several of the deceased Fellows will be detailed in memoirs, hereafter to be read.

I cannot, however, pass over in silence the promising young life of Dr. Thomas S. Kirkbride, Jr., without a special word of regret that it was cut off at the very beginning of a career which foreshadowed so much usefulness.

Moreover, it would not be proper to allow the death of Dr. Emil Fischer to pass without some notice of his unusual work at the Pennsylvania Hospital. When Dr. Fischer came to this country his means were extremely limited. He was a fine musician, and eked out a slender income, first, by teaching music, and, later, as recorded in some detail in the history of the Pennsylvania Hospital by Dr. Thomas George Morton, by cataloguing its library in 1856, when it consisted of 10,500 volumes, and was considered at that time one of the best, if not the best, collection of medical works in this country. Dr. Fischer lived in the hospital for several years while making the catalogue, and formed there delightful friendships with not a few of our older Fellows. They remember not only his refined musical taste, but the harmonious character which everyone who knew him recognized as one of his chief charms.

Very agreeable reminiscences are connected with three of the Fellows who have died during the past twelve months. Dr. Stillé left to the College library the remainder of his own medical books and made the College his residuary legatee; Dr. Ashhurst left us 1500 volumes, among which are many treasures of mediæval medical classics, and Dr. Da Costa bequeathed to us nearly 2500 volumes, many of them of unusual value from the clinical and literary point of view. In addition to this, Dr. Da Costa left a bequest of $5000 for the establishment of a Publication Fund, the income from which, as he indicated to me during his life, he hoped would prove such an addition to the annual appropriation of the College as to make possible the publication of the many valuable papers presented before the Sections of the College as well as those read at the monthly meetings of the College itself. It is evident, however, that the pub-

lication of these sectional papers would probably double the size of our volume. This would call for the annual expenditure of a sum of money requiring for its realization an investment of not less than $15,000. I hope, therefore, that some of the Fellows will be able in the course of the present year to obtain from their wealthy patients the additional amount required for this fund.

This leads me to call attention also to the infrequency of bequests to the College from deceased Fellows. Until within a few months George B. Wood, Agnew, Judson, and Da Costa are the only four Fellows who have ever bequeathed any money to the College, and Mütter and Mitchell the only two who have ever given any considerable sum of money during their lives. Books we have had from several sources, and they are always most welcome, but the larger the library the greater the need for space for housing it, the greater the expense of the cataloguing and the other running expenses of the library, and, in addition to this, beside the needs of the Publication Fund, there is looming up before the College before very long the absolute necessity of larger space. Not a few of the Fellows of the College are in such pecuniary circumstances that they can readily leave in their wills sums of money, in some cases of moderate amount, in others of large amount, and I desire especially to request the Fellows whose means allow of it, if they cannot give in their lifetime, to provide at least a bequest in money in their wills either for the Publication Fund or for the general purposes of the College without restriction. In some cases a policy of life insurance in favor of the College could secure this end in the event of inability to give larger sums during their actual lives. Moreover, it is very easy in many cases to influence wealthy patients to make bequests to the College or gifts during their lifetime. Gratitude to the individual doctor may well, therefore, find its expression in a gift to the entire profession. In soliciting this, the doctor does not render himself liable to any imputation of selfishness, but evinces rather great breadth of mind.

Less than two years ago the resources of the library consisted of: First, the appropriation from the College. This was more than absorbed in salaries and other running expenses. Second, the annual income from the Directory for Nurses. Third, the income from the

$14,000 in the various library funds. The total income from these sources was far too small for our needs, and, accordingly, after consultation with several Fellows, a determined effort was made for the purpose of raising an endowment fund of not less than $50,000. Some gifts were made conditional upon raising the entire sum by July 1, 1900. It seemed as though the effort to complete the fund on time would fail, when, through the liberality of Mrs. Wm. F. Jenks, the generous widow of a deceased Fellow, the project was brought to a successful conclusion.

Not having felt satisfied with the intellectual or scientific results of the Wm. F. Jenks Prize Fund, which she had established as a memorial of her husband, she decided very wisely to transfer this fund into the Wm. F. Jenks Library Fund. . . . .

Let me call attention to the fact that the Jenks Prize Fund—a considerable amount—has not always called forth essays of such merit that any prize has been awarded. The last time that the Hatfield Prize was to be awarded no essay worthy of the prize was presented, and a year ago when even the Gross Prize of the Philadelphia Academy of Surgery, amounting to the large sum of $1000, and which had been announced five years beforehand, was to be awarded no essay worthy of the prize was presented. With these facts before us, I think it is not inappropriate to urge, so far as the Fellows can control future gifts and bequests. that no further prize funds should be encouraged. The Library Fund, the Publication Fund, and bequests or gifts for the general purposes of the College are very much more useful.

During the last year the increase in the library, both by gift and purchase, has been exceptionally large—6294 volumes. or, excluding duplicates, 4158 volumes. It is becoming that I should express, as your executive officer, to the various Fellows and other persons, including specifically the various medical publishers of this city, who have given to our library most liberally, the thanks of the College for their generosity.

A definite system of exchanges, which now covers six foreign universities, has been established, and it is hoped will be still further extended during the coming year. Nearly 7000 theses and inaugural dissertations have been added to the library during the past year.

The need of a new card catalogue, fully up to all the modern requirements of the second most important medical library in the United States, has become so pressing that it is most gratifying that the College has authorized a special committee to collect the $2500 necessary to install this, and that the committee has already raised considerably more than one-half of this sum.

How useful the library is may be inferred from the fact that during the year of 1899 30 Fellows and during 1900 33 Fellows published new works or new editions or translations of works already published or works which they had edited. That in two years 20 per cent. of the entire Fellowship of the College should show such remarkable literary activity is most creditable.

During the year an important financial change in the policy of the College has been inaugurated, and one which has proved to be exceptionally useful even in the short period during which it has been in effect. The Pennsylvania Company for Insurances on Lives and Granting Annuities has been made the financial agent of the College. They have taken physical possession of all our securities, which are thus beyond any peril of damage or loss. They collect all rents, dividends, and interest due us, and they are the advisers of the Finance Committee in all of our investments. . . . . In no other way could we have found so easily and speedily suitable and safe investment at a reasonably remunerative rate.

In reference to the standing committees, I wish to make a suggestion to which I would ask your serious consideration. Some of our committees have consisted for a good many years of the same persons. This has its advantage in establishing a uniformity of policy on the part of each committee, and all of the members of such a committee become very familiar with their duties. It has, however, the disadvantage of excluding a large part of the Fellowship from that active participation in the work of the College which would greatly benefit us by interesting actively a much larger number of the Fellows than is now possible. In addition to this a permanent committee is apt to fall into ruts, and it is very disagreeable to suggest any alterations in the *personnel* of any committee. In order to obtain the advantages of relative permanence, and, at the same time, the advantages of gradual change, I recommend to the

College that a By-law be passed that at each annual election the senior member of the committee in length of service shall be ineligible for election for one year. I would have the change apply not only to the elective committees, but also to the committees appointed by the President. There are seven standing committees of which four—Committee on Publication, Directory for Nurses, Mütter Museum, and Finance—consist each of three Fellows; one, the Committee on Entertainments, consists of four Fellows; and two, the Library Committee and the Hall Committee, consist each of five. If the amendment proposed should be adopted four of the committees would have the possibility of a change of their *personnel* in three, one in four, and two in five years. The fact that an outgoing member of any one of these committees would be eligible to re-election after being out of office a year would insure the return of an unusually valuable member to such a committee, while the dropping of one member of each committee annually would afford an easy and unobnoxious way of changing the *personnel* of the committee.

The transfer of the Jenks Prize Fund to a Library Fund makes inoperative Article I. of Chap. XIV., relating to the Wm. F. Jenks Committee. I recommend that this article be stricken out.

I recommend that the time for the delivery of the annual address by the President be changed from December to January, for the reason that he ought to be able carefully to read over the annual reports of the Secretary, the Treasurer, and the standing committees in order to become familiar with the actual state of the College, its finances and various activities. At present, as the fiscal year ends November 30th, he cannot have over a few days, and may not have even one day in which to consult these various reports.

Turning now to the literary and scientific work of the College, one of the needs of the College is for a larger number of interesting papers. The College has only nine meetings a year. Practically very few papers can be read at the December and January meetings on account of the amount of special annual business, particularly the annual reports and the annual election. During the last year there were only eighteen papers read at these stated meetings. In order to increase the number of papers read, as well as to add to the efficiency

of our meetings, I wish to make two suggestions: First, that while no restriction shall be placed on the length of papers as published, in case any paper should exceed twenty minutes in reading that the author, instead of reading the entire paper, shall give either a written or a verbal résumé of the paper not exceeding twenty minutes in length. This will add very much to the interest of the papers. Daily fluctuations of temperature, pulse, and respiration, and other minor details, while they are important as a matter of record, are wearisome and detract from the interest of a paper very materially. If a short verbal statement of the gist of the paper is made then there will be room for that which is my second suggestion, namely, more discussion. I would propose that the author of any paper which he wishes to have discussed shall obtain the consent of one, two, or more of his friends who are willing to discuss the paper, and that these names be entered on the notices of the meeting. In a number of instances in the past year valuable papers have passed without the slightest discussion, simply because the authors had not obtained in advance the promise from Fellows to take part in such a discussion. Of course, this restriction would not apply to special papers, such as those read by invitation, but only to ordinary papers.

During the year one very valuable special meeting was held to which all the members of the Neurological Society were invited, and aided greatly in making it a success. Several distinguished medical men from New York and Baltimore were present by invitation and took part in a discussion on the fifth nerve in its neurological and surgical relations. At this single meeting there were nine papers read, one-half the number which were read during the entire year. The papers were widely published and commented upon, and the interest excited by the debate was very marked.

The exhibition also of color photography by Mr. Ives was of great value in enhancing the interest of one of the meetings about a year ago. It is proposed that Dr. Crile, of Cleveland, shall be present at a coming meeting, at which I hope we shall have a large attendance to witness his interesting demonstrations. Beside this, Dr. Wm. Osler, one of the warmest friends of the library, will ask before long for a special evening at which he will exhibit his complete set of all

the editions of the *Religio Medici*, as well as a full set of the works of Sir Thomas Browne, and will give some account of his life and writings, illustrated with lantern views. For this meeting I suggest that ladies may be invited by the Fellows.

This meeting is the last one in the nineteenth century. When we compare our present numbers with the score or less of Fellows in 1800, our permanent home, our splendid library, our many and valuable volumes of TRANSACTIONS, and our considerable invested funds, with the utter absence of all such evidences of prosperity a hundred years ago, and, in fact, even fifty years ago, we have reason to thank God and take courage. May the annual address in the year 2000 exhibit a prosperity in advance of that of 1900 even more than the prosperity of to-night exceeds that of a century ago.

THE GASTRO-INTESTINAL TRACT IN NERVOUS DISEASE.

By F. SAVARY PEARCE, M.D.,
INSTRUCTOR IN PHYSICAL DIAGNOSIS, UNIVERSITY OF PENNSYLVANIA; PHYSICIAN TO THE MEDICAL DISPENSARY, ST. AGNES' HOSPITAL.

[Read January 3, 1900.]

The intimate relation between the central nervous system, organs, and functions of the body especially cannot be too forcibly emphasized. Stress is to be laid upon the oblivion existing through the inexact knowledge of the anatomic relation between the cerebro-spinal, or system of animal life, so-called, and the ganglia sympathetic, or system of organic life, as we know it to be, and especially the inexact understanding of the anatomy of these most delicate ganglia and nerve-fibres and of their intricate inosculations—*i. e.*, the sympathetic fibres about the important organs of the body. Recent anatomical and physiological experimentation, now being pursued through the aid of vivisection, is proving more and more the wonderful rôle played by the "abdominal brain," as some anatomists have dignified the ganglia of this to be. Bearing in mind the wonderful controlling influence of the sympathetic nervous system, as shown by the preservation of life under profound anæsthesia, the justice of styling these ganglia subconscious brains is the more appreciated, and the wonderful influences even then of its reflexes upon important centres, as respiration, the writer has recently had demonstrated to him in a male, aged thirty years, whom he had operated on for fissure *in ano*. While under profound etherization the respirations could be controlled (inhibited) by wide dilatation of the sigmoid flexure by means of a long

bivalve speculum. Perhaps one great reason why the sympathetic system has not been given enough import in practical medicine and therapeutics is because morbid conditions of the same, as in health, do not give rise to any painful sensation, and, therefore, when this does occur it is directly of cerebro-spinal origin. We very often fail to seek the real reflex primogenesis of morbid states presenting symptoms for relief. In another place[1] we have emphasized the importance of considering the sympathetic system's influence in the causation of neurasthenia through disturbance of the reproductive organs—a subject that has been widely discussed and must be accepted as proven. While similarly, of course, corelation of the great laboratory of the body through the sympathetic to the cerebro-spinal axis, both physically and pathologically, needs no scientific advocate to further establish it, yet specific cases bearing upon gastro-intestinal disorders and nervous disease may prove of some value for obtaining a more wide-spread recognition and a proper therapeusis of the less manifest diseases of the nervous system which still *do* have the same pathogenesis. Admitting the difficulty of ascertaining cause or effect frequently, even where there is evident corelation between nervous disease and gastro-intestinal disorders, the point we wish to consider *especially* is to determine *how many* cases are relieved by treatment of the primary or secondary gastro-intestinal state, and more scientifically to seek out given cases of nervous disease greatly aggravated by the intestinal trouble, or, indeed, entirely caused by it. This leads back to the broad study of heredity and conditions of environment and much clinical experience. In a paper with Dr. Wharton Sinkler, on "Family Diseases,"[2] we draw conclusions that heredity could be considered from a statistical review of a large number of cases, as one or more in eighty, although, if the most accurate history of our cases could be had, much higher.

Savill (1899), in his recent monograph on *Neurasthenia*, adds zest to investigation of these allied nervous states of the human body.

[1] Neurasthenia: Its Corelation with Pathology of the Female Genital Organs, with H. D. Beyea, M.D., Annals of Gynecology and Pediatry, September, 1898.

[2] Journal of the American Medical Association, February, 1900.

Impetus in the study of aberrations of kidney excretion—not an index of renal disease, however—was first given the writer through making the urinalyses in a large number of cases of nervous diseases in the practices of Drs. S. Weir Mitchell and John K. Mitchell. The results of uric-acid findings have been published, and while indican is tested for in almost all cases we have not up to this time made careful analysis of its possible import.

The whole subject of irritation and autointoxication, *per se*, as causal factors of disease of the nervous system is most intricate in its exact determination for the therapeutic point of view, which is our *most to be desired* knowledge. For instance, in a case of large ovarian tumor, as reported by Beyea,[1] recently, to my mind, the glycosuria was due to irritation of the glycogenic centre, in turn perverting metabolism. The diabetes here disappeared after removal of the tumor.

Fourteen Detailed Cases as Basis of this Study.

Case I.—M. B., female, aged twenty-two years; married, one child living; no miscarriages; reported March 19, 1898, suffering with what she styled splitting headaches for about three months, worse toward evening and the dark hours. The headaches had begun with constipation, which, when first seen, was very troublesome. She suffered from anorexia, insomnia, and exhaustion, tongue was slimy, coated, and flabby, tooth indented, pulse 120 per minute, heart weak, no murmur. We could find no ocular or aural cause for the head symptoms. She was placed on a liquid diet, given blue mass, grains five, at bedtime for several days, and tonic of tr. nucis vomica and soda bicarb. before meals, and semi-rest treatment was enjoined. Improvement followed immediately. At the end of two months she considered herself a well woman, having no head symptoms, and the bowels remaining in normal condition.

Case II.—J. J., reported April 2, 1898; male, aged fifty-three years, shoemaker, alcoholic; following exposure awoke with pain in arms and down left leg, accompanied by numbness on the left side of the body. He never had had acute rheumatism. With the incipiency of the pain described he had had an attack of gastritis, followed by lienteric alvine dejections of gastric fermentation. Antiseptic treatment to the intestinal tract dissipated the pain and subjective numbness within twenty-four hours. Although this patient complained of dulness of vision for a week after the attack, to our mind the case presented a complex of alcoholic depression

[1] College of Physicians of Philadelphia, Section on Medicine, December, 1899.

to which was added intoxication from ptomaïne poisoning. In the history it was ascertained that the amount of alcohol he had taken previous to this attack had produced little or no impression on the nervous system. The peculiarity in this case was the objective and subjective coldness of the left arm, leg, and entire left side of trunk, probably showing disturbance of the caloric centres unilateral in the higher nervous system. At the end of two months the man seemed back to normal health in all ways. Perhaps some hemichoreic symptoms may be excited in the unstable neurons later in the course of the case.

Case III.—R. I., reported May 28, 1898; female, aged forty years; Hebrew; five children living, three dead; complained of dull vertex headache, periodic and about once in three months, lasting several days, accompanied by insomnia; no rheumatism; no other pains were complained of; bowels said to be regular; urine high-colored, *contained indican in excess;* appetite fitful. She is a plethoric woman, heart is feeble; however, no organic lesions can be found excepting great relaxation of the abdomen and dilatation of the intestinal tract with fermenting contents. The use of magnesium sulphate to thorough cleansing of the intestinal tract, with occasional use of acetanilid, caffeine, and camphor monobromate as a nerve depressant, together with massage of the abdomen and restricted diet; then occasionally high flushings of the colon with lime-water solution, produced immediate permanent improvement.

Case IV.—M. G., reported July 2, 1898; female, aged thirty-five years; single; complains of general nervousness and nerve tire, easily exhausted, with considerable vertical headache, growing worse toward evening; suffered from indigestion; eruptions of sour stomach contents; bowels are moved daily, although there is constant feeling of tightness through the abdominal contents, with distention. This woman's diet was cut down to liquids largely, and nux. and soda mixture were given before meals, with very rapid relief of general nervous state and headache.

Case V.—J. S., reported August 6, 1898; male, aged twenty-nine years; no history of nervous disease in the family; complains of pain in the head in the occipital region, lasting one week, completely incapacitating him from work. There is no history of specific disease; kidneys are normal, his physique being powerful. Gave a history of extreme constipation for some months, and was a great meat eater. The only pathological state of the urine was of *excessive indican.* He was put on restricted diet and given sodium salicylate, gr. x, t. i. d. Within a month this man got perfectly well of the toxæmic headache, considered to be due to ptomaïne intoxication aggravated by excessive proteid diet.

Case VI.—C. K., reported December 3, 1898; female, aged thirty-nine years; married, eleven children, six living, others died of marasmus. Patient complains of frontal headache for the past two weeks, which comes on in the morning and disappears in the afternoon; also complains of

vague pains in extremities and of *general nervousness*. Headaches begin about 9 A.M. and last to 4 P.M. Correcting a hyperacidity of the stomach with mild daily catharsis very much relieved her condition at the end of four weeks—that is, the headaches did not last so long, but seemed quite as severe in their several hours' duration. This woman seemed entirely well two months after beginning treatment. During the summer of 1898 she relapsed into the same condition of intestinal indigestion with *indicanuria*, to again be entirely relieved in the fall by a mixture of charcoal, gentian, and sodium bromide. Again having had a relapse, living as she does in poor hygienic surroundings, each time has been relieved by intestinal antiseptics. It should be said there were no other causes to be found; menstruation was normal, and the eyes presented no error of refraction.

CASE VII.—N. D., reported March 25, 1899; widow, aged thirty years; two children living and well; no miscarriages; has been very constipated for some weeks; her head began to distress her much across the frontal region, beginning several weeks before reporting for treatment. Pain is of dull nature, gradually growing worse toward evening. This patient was much improved by the use of bromide and gentian, and after full correction of intestinal torpidity the headaches entirely disappeared. I take it that the absorption of ptomaïnes from the intestinal tract and of the improperly eliminated metabolites of the system passing through the sensitive dura mater were the exciting cause of the cephalic distress.

CASE VIII.—M. P., reported April 11, 1899; female, aged sixty-five years; complaining of pain in the lower lumbar region and general feeling of tire, accompanied by violent wind dyspepsia, and labelled "disturbed metabolism." Under the use of gentian, nux., and soda mixture with the limitation of diet to milk and proteids, and with the stimulating effect of the static current down the spine, she was entirely relieved in two and a half months of the general toxæmic state. *The association of dyspepsia and rheumatism* seems to be proven in such a hybrid case as this one. We have no doubt that many cases of rheumatism are precipitated by catching cold, since the retention theory of the genesis of a "cold" would tend to show the disturbance of metabolism acting through the nervous system, and would be sufficient cause for the rheumatic state. That some form of organism may be a determining factor of acute rheumatic fever, and may, also, be a potent cause, certainly the subject of rheumatism is still in a state of theoretic speculation. Nervous origin is, undoubtedly, a large basic cause in this disease, so closely allied to the conditions of waste product pains of disturbed metabolism; for the nervous system must control the proper absorption and elimination of lucomaïnes, etc., in health and disease.

CASE IX.—L. L. (private), reported May 11, 1898; female, aged thirty-five years; for three years has been suffering from excessive nervousness associated with extreme constipation, bloating of the stomach, and wind

dyspepsia; urine contained a large amount of *indican;* her sleep was much disturbed, and vague distress in the head much aggravated when bowels were not relieved by cathartics. Use of bromide of soda in moderate doses, with trional as a hypnotic, much improved her general nervousness. But it was not until full relief of the gastro-intestinal condition, after several months in treatment by means of regulated diet, massage to the abdomen, special as well as general, and the use of salol as an intestinal antiseptic, she began to gain flesh and lose permanently the general nervous condition she had been in so long. The difficulty in such a case as this is to trace the *post hoc* in the vicious circle undoubtedly set up; and yet we feel that clinically restoration of health was largely influenced by correcting the function of the digestive tract.

Case X.—F. S., reported to the Howard Hospital service of Dr. J. Madison Taylor, July 14, 1897; female, aged seventeen years, single, came to us at the nervous clinic, having been referred from the medical clinic on account of what was diagnosed as *toxæmic headaches*, the ordinary methods of relief having failed. As the girl came into the room she gave a peculiar sigh due to a forced inspiration, and fell on a chair *unconscious*, frothing at the mouth, and clonic and then tonic spasm of the entire musculature. She was *profoundly unconscious* and pallid in the attack, and was dazed for half an hour after recovery. It was then learned she had had *five* of these seizures within a year. She was particularly waxy in appearance, and there was acne over the forehead and nose. She complained almost constantly of frontal headaches, was extremely constipated, and suffered much from epigastric distress, although the trio of signs of epigastric ulcer were eliminated. This girl complained of tenesmus and general abdominal distress much increased at the time of attempted defecation. The skin eruption seemed to be aggravated and to spread as to more generalized hyperæmia about 10 o'clock in the mornings, increasing to acne about 3 o'clock, when small white vesicles came out over the face and neck, this more acute syndrome of epidermic disorder again abating toward night. The patient was first put on a tonic, and the case studied carefully from day to day. It was soon found she complained of general muscular soreness without any renal rheumatism. We now put her on small doses of potassium iodide and sodium salicylate, and began to pay special attention to the bowel condition, as examination of the urine revealed large quantities of *indican* present. High enemas were resorted to, by the use of the long rectal tube, adding glycerin to the alkaline solution of soap. It was *four* months before any approaching normality of the intestinal tract was obtained, and with it the appetite improved, persistent acne was bettered, while the transient erythrodermia had entirely disappeared. The patient was now put upon iron in moderate doses (Blaud's pill, 5 gr., t. i. d.), and the various laxatives used, as the great torpidity required frequent changes of cathartics to produce any effective results. It should be said no stricture of

the lower intestine could be made out. While the girl complained constantly of stomach distress, since the headaches were better and no eye lesions or other cause having been found for them, we felt we were on the right track. During the following year the patient had but one epileptic seizure; the blood-count ran up from the original anæmic condition of 3,200,000 red blood-corpuscles to 4,800,000 red blood-corpuscles, and while iron could not be taken for many weeks at a time the ability to digest food through the use of carotid and other digestants favored hæmogenesis. The patient has had no epileptic seizure for *twenty-two months*, and seems at the present writing to be about well, so much so that she was married on December 13, 1899. The urine analysis failed to detect indican at the present time, and the gastro-intestinal function is entirely normal.

Case XI.—A. L., single, aged fifty-two years, always a delicate, refined woman, was sent to me by Dr. Stewart, of Allegheny, January, 1899, suffering from tormenting headaches not of a migraine type, but being constant, dull, and splitting in character all over the cranium. She has been violently constipated for twenty years, but paid little or no attention to it; doing with little food; having generalized swelling of the face, hands, and lips at times in the mornings when she was at her work. The urine was normal save for much indican present. She frequently "threw off" quantities of bile. There is some tenderness over the liver, and gastric irritability with gastroptosis. She is in extreme neurasthenia. Climacteric established at fifty years of age, but did not seem to influence headaches, constipation, nor her generally poor metabolism. She was placed on high enemas, free use of cascara and abdominal and general massage, occasional lavage, and trophomine as a tissue builder. Improvement was slow but steady. Her eyes were *not in any way causative of headaches*, being simply presbyopic and fully corrected. After six months she had become very well indeed. I saw her in Allegheny on December 22, 1899, after a two days' headache, apparently precipitated by constipation, with high pulse tension, against which we are with some success treating. The use of molasses in this case will precipitate headaches the next day, due, I take it, to fermentation thus set up. (Since the above report she is much improved.)

Case XII.—C. S., aged fifty years, business man with large responsibilities for years, with an original weak constitution; nervous. Has had gastritis off and on for twenty-five years. Has been treated with great benefit on several occasions by the late Dr. William Pepper and by Dr. Wharton Sinkler, the latter gentleman placing the patient on semi-rest treatment, sulphocarbolate of zinc and the exhilarating effect of hydrotherapeutics at the Infirmary for Nervous Diseases. At this time the *total acidity* of the stomach was 4 *per cent.*, and showing slow digestion in the lavage fluid drawn off two and one-quarter hours after a test-meal. There was no albumin or glucose in the urine, but an excess of uric-acid crystals. The man also had irritating piles, removed by the surgeon. His bowels became regular,

and abdominal distress largely disappeared. He gained much in weight, and after a number of weeks' treatment went back to Ohio considerably improved, May, 1899. While in Ohio, December 24th, I was called to see him; found all signs of *returning neurasthenia, gastric atony, and indicanuria.* He had lost much flesh. Was belching continuously. Attack said to be due to cold. Blistering over stomach was now resorted to, and the toxic condition of the urine treated by salol. The patient has improved some up to date, and indicanuria has disappeared. Undoubtedly in this case auto-intoxication plays a rôle in semeiology.

Case XIII. is that of a female, aged seventeen years, white, single, suffering one year from atonic dyspepsia and persistent *supraorbital neuralgia* for six months. She is of bilious temperament. Cardiac arrhythmia prominent. Gastric antisepsis cured the neuralgia in one month.

Case XIV.—Anna G., German, single, aged twenty-six years, reported to my clinic at St. Agnes' Hospital, November 9, 1899, suffering from constipation since sixteen years of age, *frontal headache, acne, and lethargy.* The latter two symptoms were always worse in the morning, and recovered some from them as she forced herself out-of-doors or to her work. Is always worse after a hearty meal, when she has a sense of sinking. Bowels are moved daily, but she has a sense of insufficient evacuation always. Stomach is *dilated* down to the umbilicus. *Indicanuria marked.* Indigestion is always made worse by worry or overwork. If she catches cold the bowels are always made more costive. Boulimia and evidences of slight catarrhal gastritis. She was put on restricted diet, given sodium phosphate, ʒj, before breakfast, acid nitrohydrochlor. dil., gtt. x, a. e., with a tonic. This girl has very rapidly convalesced from her miserable condition, and is happy in the zest for work.

A sister is undergoing such a therapeutic *régimé* with good effect.

This case shows the mixed class we have styled—*i. e.*, when neurasthenia and auto-intoxication exist contemporaneously. The combined neural upbuilding and intestinal antisepsis and hygiene we feel are equally responsible for the great improvement up to date. Such a case as this might be termed one for the neurologist or for the gastrologist, if we do not look too broadly on the medical horizon.

Conclusions. From the foregoing clinical study it seems pretty clearly demonstrated to the writer that we have three classes of disease in which the nervous system is more or less intimately influenced by gastro-intestinal disorder and usually of a toxic nature.

1. (*a*) Neurasthenic states, general or in localized areas of distribution (most common); (*b*) where organic changes are present in the nerve cells (sclerosis). Both of these influences certainly lower resisting power and cause improper distribution of nerve force to the gastro-intestinal tract. This leads to dilatation and perversion of the enteric secretions, thus giving rise to fermentations of the stomach contents, etc., products of which are reabsorbed, further disturbing the metabolism and aggravating the symptoms of neural disorder.

2. Cases where long-continued gastro-enteritis causes organic change in the mucosa and gastro-intestinal glands, including the liver and pancreas, of course—thus making the primal point of disease an irritating fermentation, with the elaborations of toxins. These, with the hyperacid secretions, especially of the stomach contents, are in part absorbed into the system, producing a constant auto-intoxication, as would excessive tobacco or any other poison, thus intoxicating through the blood the central nervous system until we have finally, in slow process, asthenia set up in the neurons, innervation of the gastro-intestinal tract at that moment being perverted through the efferent, specialized, and trophic nerve twigs.

3. There must be a mixed class of cases in which neurasthenia, so-called, or organic nervous diseases are associated, *pari passu*, with disorders of digestion of a functional or organic nature. Admitting the difficulty of determining when *one* and *two* exist—even when the cause and effect certainly do maintain—for this reason it seems logical to state that a large number of cases placed in the *third* category belong by right to the preceding *two* subdivisions. The more close histories of our cases procured, the more exact clinical studies made of symptoms and signs in the individual case, together with careful analyses of the secretions and excretions of the body, thus utilizing the associated import of such phenomena as *indicanuria* for indices, the better will be the results obtained in *treatment*. So, also, will such closer observation in the direction noted more surely place the association of neural and somatic diseases out of the less intricate classification we have termed *mixed*. The individual case study as to hereditary diathesis or tempera-

ment must needs be broadly taken into account. There does seem to be the greatest reason to assume the *acid*, *neutral*, and *alkaline* temperaments with their predispositions and peculiar immunities in drawing any conclusions of the case.

An admirable article by Dr. A. E. Sterne, in recent numbers of the *Philadelphia Medical Journal*, elaborates this, which seems to us a very important side of medicine. Nor should we fail to recognize the secretions and excretions as influenced by the emotions—*e. g.*, serous diarrhœa produced through emotion or overwork, as shown in the novitiate to the examiner's room, or application to hard mental labor in a neurasthenic subject. These physiological phenomena are shown in full light by the work of Darwin on *Expression of the Emotions in Man and the Lower Animals.* Hence the subtle metaphysical influence must be weighed in any case along with the accurate scientific knowledge indicated.

The more we study the sympathetic nervous system the more clearly will morbid phenomena, such as the association of biliary disease or irritation causing disturbance of menstruation, of serous diarrhœa substituting menstruation, or, indeed, vicarious menstruation, be better understood. The fact that pain seldom enters into these disturbances as a prominent factor at least misguides the patient as to interpreting his being ill, and the physician, too, in determining the conditions from the limited objective signs and symptoms alone.

Addendum. Aside from the subject of intoxication, of course other irritants of a mechanical nature do produce nervous symptoms. I know of a case in charge of a Western physician where chronic eczema was cured by stretching a stricture of the rectum, and another case of epileptic convulsions ceasing after a cure of a rectal fissure which had been long neglected.

A similar case has been recently reported in the *Boston Medical and Surgical Journal*, December, 1899. The writer wishes here to refer also to the *New York Medical Journal* for July 29, 1899, in which ("Odd Types of Disease") he has reported a case of hebephrenia, with all its typical vagaries, undoubtedly due largely to persistent constipation. The girl is perfectly well to-day.

SOME OLD CERTIFICATES OF PROFICIENCY IN MEDICINE.

By FRANCIS R. PACKARD, M.D.

[Read January 3, 1900.]

Last November Dr. Keen secured for the College of Physicians, through the kindness of Julius F. Sachse, Esq., a copy of a certificate granted by Dr. Christopher Witt to one of his pupils. The original is in the collection of Judge Pennypacker, of this city, who generously loaned it for this purpose. Dr. Keen's copy is presented to the college this evening.

In the certificate Dr. Witt testifies to his pupil having received instruction, not only in mere medical science, as we understand it, but also in the astral sciences, thereby giving him a great advantage over those who had received their medical education at the hands of physicians who were not proficient in the science of the stars. There is an interesting account of Dr. Witt in Mr. Sachse's book, *The German Pietists of Provincial Pennsylvania*. He was a quaint character, even for his times, as we look back on them. Coming to this country in 1704, he settled first with the German pietists on the banks of the Wissahickon, but in 1718 moved to Germantown. There he remained until his death, in 1765, becoming distinguished as a physician, naturalist, astronomer, and magus. He was also an expert clockmaker and builder of pipe organs. His astronomical investigations were made through a large telescope in his house, and these occult studies earned for him the name "hexen-meister," or master of the cobolds or fairies. He had a mulatto servant whom the neighbors regarded as his famulus, or familiar spirit. He was an intimate friend of John Bartram, the

botanist, and a correspondent of Peter Collinson, the famous English botanist. At his death he bequeathed £60 to the Pennsylvania Hospital.

In the days before there were any medical colleges to grant diplomas, certificates such as this were all that many practitioners of medicine could show to indicate any special fitness for their calling.

It has been estimated that at the outset of the war for independence there were upward of 3500 persons practising medicine and calling themselves physicians in the colonies of whom not more than 400 had received the degree of M.D., or even B.M.

Wickes[1] furnishes us with a copy of a certificate issued by Dr. John Redman:

"Medical Certificate to Mr. Samuel Treat, 1765."

Philadelphia. This is to certify all whom it may concern that Mr. Samuel Treat hath served as an Apprentice to me for nearly four years, during which he was constantly employed in the practice of Physic and Surgery under my care, not only in my private business, but in the Pennsylvania Hospital in which character he always behaved with great Fidelity and Industry. In Testimony of which, I have hereunto set my hand this first day of September one thousand seven hundred and sixty-five.

Signed John Redman.

We whose names are under written do Certify that Mr. Samuel Treat hath diligently attended the practice of Physic and Surgery in the Pennsylvania Hospital for several years.

Signed Thos. Cadwallader,
Phineas Bond,
Th. Bond,
Wm. Shippen,
C. Evans.

This is to certify that Samuel Treat hath attended a course of Anatomical Lectures with the greatest diligence and assiduity.

Signed William Shippen, Jr.

Dr. Thomas G. Morton, in his history of the Pennsylvania Hospital, also gives some interesting specimens of the certificates granted by the hospital authorities to those who had attended at

[1] History of Medicine in New Jersey.

the hospital as pupils. They were issued after the following form:

"This is to certify that ******** son of ******* West Jersey, entered regularly as a pupil of the Pennsylvania Hospital **** 1763 and continued his attendance with diligence and application, to **** 1764 during which time we hope and have reason to believe he has made considerable Progress in the Knowledge of Anatomy and the Practice of Physick and Surgery, therefore wishing Happiness and success we give from under our hands and the seal of the Corporation, this Testimonial of our Esteem and Approbation."

The first man to receive a medical diploma in North America was Daniel Turner, who received the gift of an honorary degree of Doctor of Medicine in 1720 from Yale College. Turner had given much money to the college, and the degree was intended as some return for his generosity, hence those of a humorous turn of mind are said to have interpreted M.D. as signifying Multum Donavit.[1] There was, however, no medical department at Yale until the year 1813.

At the centennial celebration of the Medical and Chirurgical Faculty of Maryland, held in Baltimore in April, 1899, there was exhibited the medical diploma of Dr. Archer, from the College of Philadelphia. This is probably the first medical diploma awarded after a course of study in America. As such it merits reproduction. It will be observed that the Faculty signatures comprise the eminent names of Morgan, Kuhn, Bond, and Shippen, probably the four most prominent medical men of their day in this country.

Omnibus ad quos praesentes Literae pervenerint, Salutem. Nos Praefectus, Vice Praefectus, et Professores Collegii et Academiae Philadelphiensis, testamur virum ornatum ac ingenum Johannem Archer assidue interfuisse, operamque sedulo navasse, ut Scientia Medica imbutus atque eruditus discederet, ac postquam, Curriculi sui Spatio peracto, in Aula nostra coram Curatoribus, multisque aliis Civibus dignissimis ad Examen revocatus, se in omnibus hisce Studiis satis versatum comprobasset, ex Curatorum Mandato in Publicis Comitiis vigilissimo primo die Junii Anno 1768 celebratis Baecalaureatus in Medicina Gradum, omniaque Privilegia, et Honores ad hunc Gradum pertinentes consecutum fuisse. In cujus Rei Testimonium

[1] Toner. Annals of Medical Progress.

his Liberis, majori Collegii et Academiae Sigillo munitis, Die Annoque praedictis Nomina subscripsimus.

Johannem Morgan, M. D., F. R. S.,
Theo. & Prax. Med. Professor.
Adam Kuhn, M. D.,
Mat. Med. & Bot. Professor.
Gul. Smith,
Collegii & Acadae Praefectus, S. T. D.
Fra. Alison, S. T. D. Coll.,
Vice-Praefect. & Acadae. Rector.
Eben Kennersley,
Ling. Angl. & Orat. Prof.
Gul. Shippen, M. D.,
Anat. Prof.
Jas. Davidson,
Ling. Graec. & Lat. Prof.

Fidem facio Virum ornatum Johannem Archer, Praelectionibus Clinicus et Praxeos Noscomio Philadelphiensi interfuisse et Fructus Diligentiae suae uberrimus consecutum fuisse.

Th. Bond (A. M.?),
Collegii et Acad. Curator & Praelec. Clinicus.

The earliest law, and the only one passed until many generations later, that I can find making any distinction between those who had received a diploma in medicine and those who had no degree was "An Act for Regulating the Fees and Accounts of the Practicers of Physic," passed by the Virginia Assembly in 1636. "Surgeons and apothecaries who have served an apprenticeship to those trades" were to charge at one rate, and "those persons who have studied physic in any university, and taken any degree therein," were permitted to charge at a higher rate.

In 1758, William Smith, in his *History of New York*,[1] wrote: "A few physicians among us are eminent for their skill. Quacks abound like locusts in Egypt, and too many have been recommended to a full practice and profitable subsistence; this is less to be wondered at, as the profession is under no kind of regulation. Loud as the call is, to our shame be it remembered, we have no law to protect the lives of the king's subjects from the malpractice

[1] Quoted by Canon. History of the Medical Department of the University of Pennsylvania, p. 23.

of pretenders. Any man, at his pleasure, sets up for physician, apothecary, and chirurgeon. No candidates are either examined, licensed, or were sworn to fair practice."

DISCUSSION.

Dr. A. C. Abbott: It may be of interest to the hearers of this paper to know that the Medico-Chirurgical Faculty of Maryland has in its possession not only the diploma referred to by Dr. Packard in his interesting paper, which, as he has stated, is the first medical diploma issued by the University of Pennsylvania, but it has also a portrait of Dr. John Archer, the recipient of that diploma. Both of these treasures were presented to the Medico-Chirurgical Faculty of Maryland by the surviving members of the Archer family. For some time we, at the University, have been making an unofficial effort to secure one or the other of these valuable possessions, believing as we do that since Dr. Archer was the first individual graduated in medicine by the University, we might reasonably lay claim to something tangible that would remind us of this fact. Thus far we have secured neither the diploma nor the portrait. I make these remarks for the purpose of inducing anyone who may be in this audience and who may have influence either upon the Maryland Faculty or upon the Archer family of Maryland to use it in favor of the University in this matter.

Dr. W. W. Keen: I have no doubt that the College of Physicians would be very glad to receive either the portrait or the diploma of Dr. Archer.

MEDICAL SOCIETIES IN THIS COUNTRY FOUNDED PRIOR TO THE YEAR 1787.

By FRANCIS R. PACKARD, M.D.

[Read January 3, 1900.]

THROUGH the efforts of Drs. S. Weir Mitchell and Ewing Jordan there has been secured for the College of Physicians the original diploma of Dr. William Martin, granted to him by the American Medical Society of Philadelphia in 1786. Dr. Keen very kindly suggested that it would be interesting in this connection to have read to the College a *résumé* of the few facts which are known concerning the earlier medical societies, with a view of establishing their chronological sequence.

I shall number the societies according to the order in which they were founded.

1. From 1735 until at least 1741 there was a medical society in Boston. Its existence has been doubted, but the following facts prove it conclusively to have not only existed but flourished. On February 18, 1735, Dr. William Douglass, of Boston, wrote to Dr. Cadwallader Colden, of New York:[1] "We have lately in Boston formed a medical society, of which this gentleman (Dr. Clark), a member thereof, can give you a particular account. We design from time to time to publish some short pieces; there is now ready for the press number one, with this title-page:

Number One.

Medical Memoirs.

Containing:

"1. A Miscellany. Practical introduction.

"2. A history of the dysentery epidemical in Boston.

[1] Massa. Hist. Soc. Coll., 4th series, vol. ii.

"3. Some account of a gutta serena in a young woman.

"4. The anatomical inspection of a spina ventosa in the vertebræ of the loins in a young woman.

"5. Some practical comments or remarks on the writings of Dr. Thomas Sydenham.

"Published by a medical society in Boston, New England."

This title-page indicates a most interesting number, but, unfortunately, for some unknown reason, it was never published.

In 1763, Dr. Douglass published a pamphlet on *The Practical History of a New Epidemical Eruptive Miliary Fever, with an Angina Ulcusculosa which Prevailed in Boston, New England, in the Years* 1735 *and* 1736, which he dedicated "To a Medical Society in Boston." The preface began as follows:

"Gentlemen: This piece of medical history does naturally address itself to you, considering that I have the pleasure of being one of your number, that you have been fellow-laborers in the management of this distemper, and therefore competent judges of this performance, and that where difficult or extraordinary cases have occurred in any of your private practice I was favored to visit the patients in order to make a minute clinical inquiry—in short, without your assistance this piece would have been less perfect and not so well vouched."

Dr. S. A. Green[1] mentions a long communication in the Boston *Weekly News-letter* for January 5, 1737, addressed "To the Judicious and Learned President and Members of the Medical Society in Boston," and signed Philanthropos. It was a plan for the regulation by law of the practice of medicine in Massachusetts. He also mentions that the same newspaper for November 13, 1741, contains a report of an operation for stone in the bladder on Joseph Baker, aged six years. It was performed "in presence of the medical society" by Dr. Sylvester Gardiner, a well-known Boston physician. It began: "A medical society in Boston, New England, with no quackish view, as is the manner of some, but for the comfort and benefit of the unhappy and miserable sufferers by the excruciating pain occasioned by a stone in the bladder, do publish the following case."

[1] Centennial Address before the Massachusetts Medical Society, 1881

2. Wickes[1] found in the library of the New York Academy of Medicine a manuscript with the following title: "An Essay on the Nature of ye malignant Pleurisy that proved so remarkably fatal to the Inhabitants of Huntington, Long Island, and some other places on Long Island, in the winter of the year 1749. Drawn up at the request of a Weekly Society of Gentlemen in New York, and addressed to them at one of their meetings," by Dr. John Bard, of New York, and as the manuscript bears the date 1749 and the writer speaks of the epidemic as now prevailing, it is to be inferred that the society existed in that year. I know of no other mention of it.

3. In 1765 a number of medical men in Philadelphia formed themselves into a society under the name of the "Philadelphia Medical Society."

It enjoyed a brief existence of three years, at the expiration of which period it merged with the American Society for Promoting Useful Knowledge, which subsequently changed its name to the American Philosophical Society, and as such is probably the best known scientific society in America at the present day. When the societies united the members of the Philadelphia Medical Society were Drs. Graeme, Cadwallader, Redman, Morgan, Kearsley, Clarkson, Bayard, Harris, Rush, Sowman, Glentworth, and Potts. None of its records have descended to us.

4. The oldest of still-existing medical societies is the Medical Society of New Jersey, which was organized in 1766, and has maintained a continuous existence ever since.

5. Wickes[2] quotes from Dr. Peter Middleton's "Introductory Lecture at the Opening of the Medical School in King's College," in November, 1769, his mention of the "institution of societies," or "well-regulated associations of gentlemen" for the advancement of the profession, and the doctor's remark, "And permit me to add as one of the many instances of the utility of these societies that whatever merit there is in the present institution it was first planned and concluded upon in a medical society now subsisting in this place, and may it long subsist." It would appear, however,

[1] History of Medicine in New Jersey. [2] Loc. cit.

that the worthy doctor's wish was not fulfilled, as we know nothing further of the society to which he referred. Wickes supposed this society to be identical with the one which, as we have seen, existed in New York in 1749 ; but it seems hardly possible that a society which amounted to anything could allow twenty years to elapse without giving any evidence of vitality.

6. In the *Columbian Magazine* for April, 1790, is an account of the American Medical Society, written by its secretary and published by order of the society. It was instituted in 1773 by the teachers and students of medicine in Philadelphia. It went out of existence about the year 1792. A diploma granted by it to Dr. William Martin has been presented by one of his descendants to the college, and is exhibited to the Fellows this evening.

7. On May 14, 1780, a meeting of physicians was held in Boston, which organized the Boston Medical Society. Chief among its founders were Samuel Danforth, Isaac Reed, Jr., Thomas Kast, and John Warren. The object of the society was to regulate physicians' fees. The war, then drawing to a close, had upset all business relations, and the high prices of the necessaries of life, as well as the depreciated currency then in circulation, required a change in the modes of payment which had prevailed before the war. Most of the founders of this society were also prominent in establishing the Massachusetts Medical Society.

8. The Massachusetts Medical Society was founded in 1781, and is still flourishing.

9. As early as 1763 a number of physicians of Norwich, Connecticut, had petitioned the General Assembly of the State to permit them to form a society for the purpose of regulating the practice of medicine, but the Assembly refused their petition. In 1774 and again in 1779 attempts were made to organize the medical men of the State, with a view to regulating medical practice. It was not, however, until 1783 that any permanent organization of physicians was established in Connecticut. On December 10, 1783, Drs. Leverett Hubbard, Eneas Munson, Samuel Nesbitt, Levi Ives, and Samuel Darling, of New Haven, published an advertisement in the Connecticut *Journal* calling a meeting of the medical men of New Haven County at the Coffee House in New Haven

for January 5, 1784, "in order to form regulations within the line of their profession of the utmost importance to the public and themselves." Thus was the Medical Society of New Haven County founded. In 1788 it published the first volume of medical transactions ever issued in this country, a copy of which is now in the library of this College. The New Haven Society, as soon as it was instituted, sought to promote the formation of similar societies in other counties of Connecticut, with a view to the ultimate formation of a State Medical Society. This effort met with slight response. A New London Medical Society was formed, but it does not appear to have led an active existence.

10. In 1787 was founded the College of Physicians of Philadelphia, the history of which is to be found written in the fullest and most entertaining manner in Dr. Ruschenberger's *History of the College of Physicians of Philadelphia.*

We thus see that prior to the founding of the College of Physicians of this city, in 1787, there were nine medical societies which at one time or another led a more or less active existence. Of the nine there are but three that survive to the present time—the Medical Society of New Jersey, the Massachusetts Medical Society, and the New Haven Medical Society—so that our honored College may justly claim to be the fourth in seniority of the medical societies of this country.

DISCUSSION.

DR. S. WEIR MITCHELL: I was under the impression that the Medical Society of New Haven was a State Society. When I wrote my Centennial Address for our College I considered this matter and came to the conclusion that the College of Physicians was the oldest of the medical societies which was not a State or Colonial organization; these latter had usually some definite purpose which was connected with giving permission to practice.

I have been much pleased to see the younger men of the College turning their attention to the history of our profession in this country. I have been much interested in Dr. Packard's paper.

A LANTERN DEMONSTRATION,

Illustrating the Chief Points in a Research which has been Conducted for the Last Four Years Upon the Vascularization of the Ovary and Its Bearing Upon the Differentiation of Sex, Origin, Development, and Degeneration of the Graafian Follicle; Inauguration of Ovulation; Organization and Retrogression of the Corpus Luteum, and the Final Cessation of Ovulation.

By JOHN G. CLARK, M.D.

[Exhibited January 3, 1900.]

This investigation was originally begun in the thought that it would be comparatively simple to determine the normal distribution of the arteries and veins of the ovary and their relationship to each other. In the course of the study of serial sections of a few injected adult ovaries, Dr. Clark was at once convinced of the futility of attempting to draw any conclusion from this source, for the close crowding together of the parallel vessels of the medullary portion and the markedly irregular course of those in the cortex, or follicle-bearing zone, rendered impossible any accurate observations concerning the relative number and distribution of the veins and arteries and the exact course followed by each system.

With a view, therefore, of securing ovaries possessing a simpler scheme a study was made of the lower animals, such as the dog, rabbit, guinea-pig, sheep, and pig, but with unsatisfactory results, and only after the injection of the generative organs of a monkey was a suggestive clue secured. Beyond this point, however, it was

difficult to proceed, and only after the injection of a very large series of ovaries from individuals, ranging in age from a six months' fœtus to a woman many years beyond the menopause, were final conclusions reached.

In the search for this normal scheme through an extensive number of serial sections, various questions directly dependent upon the circulation presented themselves for solution, which widened the scope of this work until it developed into a composite anatomical and physiological research.

Thus the various phenomena have been considered which transpire within the follicle from its embryological origin and progressive growth to the time of its disappearance either through an obliterative process or through its rupture, organization as a corpus luteum, and final retrogression as a corpus fibrosum. In this connection Dr. Clark has advanced theories as to the cause of ovulation, the synchronism of ovulation and menstruation, the mechanism of the rupture of the mature follicle, and the final cessation of ovulation, which have been based upon observations made in the study of a very large number of sections.

Soon after beginning this work he was struck not only with the difficulty of arriving at a definite knowledge of the scheme, but also of determining the age at which this scheme may be taken as a standard for comparison.

This is certainly not possible after active ovulation is established, for the constant changes in the vascular system induced through the maturation, rupture, and organization of the follicle introduce an element of variability into the circulation of this organ which occurs in no other.

Failing to reach any satisfactory starting-point in the adult, the ovary of a girl approaching puberty was next studied by Dr. Clark, but with little less success, for it was found that almost as constant variations occur in the follicular circulation before as after the inauguration of ovulation. In the hope of finally reaching a period in the life of the female individual at which a definite standard for comparison might be found, numerous specimens from children of various ages were injected and closely studied. Finally the ovary of a six months' fœtus was obtained, which furnished a definite clue

as to the arrangement of the vessels, but as the follicular apparatus was still in process of development, a new-born child in which the tunica albuginea was well formed was selected as the standard. Even here the solution of the question was not easy, for in order to trace the ramifications of the vessels from the point of their entrance into the ovary to their ultimate termini the study of the serial sections of many ovaries was necessary.

In the embryological considerations of this subject an explanation of the origin of the spermatic vessels as an independent system from the primitive circulation of the Wolffian body was offered. As is well known among embryologists, the Wolffian and Müllerian ducts are well formed, and the germinal eminence is of considerable size before visible signs of the differentiation of sex become manifest. Up to this point the embryo is said to be of the hermaphroditic or indifferent type.

In retracing the steps of development from the well-formed embryo back to this period, some very interesting points concerning the differentiation of sex have been secured. Dr. Clark called attention to the fact that the radical differences existing between the vascular system of the testicle and ovary furnish a valuable sign for determining the gender of very young embryos before the external differential marks are established.

The fact to which attention was directed especially is that the testicular circulation is peripheral, the main artery of which courses over the dorsal aspect of the organ, giving off in its course rib-like branches which in turn send penetrating branches into the gland. Between the arteries are situated the collecting veins which unite at the base of the testicle to form the spermatic plexus.

In the ovary this scheme is exactly reversed, the arteries with their accompanying veins entering the centre of the organ, where they branch tree-like and terminate as a fine capillary anastomosis in the tunica albuginea.

Upon the peculiarities of each circulation Dr. Clark bases the differential signs of sex—a visible dorsal vessel always indicating a male, and an alabaster-like, non-vascular white cortex a female embryo.

In microscopical sections the presence of large peripheral vessels also indicates the male, whereas large central vessels indicate the female sex.

The significance of the vascular arrangements in the testicle and ovary was also discussed from the physiological stand-point, and to the radical differences existing between them were ascribed the persistence of the testicular function in the male to old age, and the comparatively early abrogation of ovulation in the female. In the testicle the production of sperma is a more or less fixed and constant function, like that of the pancreas, the parotid, and other secreting glands; consequently the circulation is not subject to variations, and is only interrupted through disease or through senile changes, whereas in the ovary there is a constant variation in the circulation incident to the obliteration or disappearance of follicles and the compensatory production of connective tissue which sooner or later begins to limit the peripheral circulation, and this in turn leads through secondary influences to a final cessation of ovulation.

The speaker remarked that these wide differences in the circulation led him to the conclusion that the origins of the ovary and testicle are not, as generally believed, the same, but are totally different, and that the expressions "a sexual period," "hermaphroditic stage of the embryo," etc., merely serve to mask our inability to select the differential features of the sexes back of this point.

In view of the fact that the common progenitor of the ovary and testicle is the Wolffian body, and that the atrophy or degeneration of the latter is coincident with the active growth of the former, Dr. Clark has endeavored to discover the explanation of this apparent paradox. According to his observations upon this point, it lies in the fact that the vascular system of the sexual glands originates entirely independently of that of the Wolffian body, consequently the synchronous development and degeneration of the two sets of organs is possible.

Having traced the development of the circulation in the ovary and testicle from the so-called asexual period to the point where they have formed systems diametrically opposite in their distribution and ultimate arrangement, the further consideration of the

testicle was dropped and the study of the ovary along the line of its development and progression to its ultimate history was pursued.

In considering the development of the Graafian follicle, Dr. Clark said that his study had led him to reject the Valentine-Pflüger theory concerning the origin of the follicle, and to accept with some reservation the general scheme of development as suggested by Waldeyer. So far as the genesis of the "egg nests" and their ultimate subdivision into follicles are concerned, he is in accord with the latter investigator; but as to the origin of the so-called follicle epithelium or membrana granulosa, he feels that the evidence in his hands is sufficient to put him at variance with Waldeyer's conception, and to incline him toward that of Foulis, who believed that the germinal epithelium only forms ova and that the lining memebrane of the follicle is derived from the connective tissue stroma.

With the completion of the fibrous covering of the ovary (tunica albuginea) shortly after birth, the vascular system becomes fully developed, and this period, therefore, is taken by Dr. Clark to represent the typical scheme, for up to this point there has been no derangement of its central or peripheral branches, which will occur later through the progressive development and degeneration of follicles.

The secondary branches of the circulatory tree occupy a comparatively small medullary area, its tertiary branches being given directly off into the follicle-bearing zone. The follicles are, as a rule, still in their primitive state, only a few of the many thousands as yet showing progressive development.

Even at this early period, however, isolated follicles undergoing progressive and retrogressive changes may be noted. These changes, according to the speaker, are closely analogous to if not identical with those occurring in the ovaries of older children and in women after ovulation is inaugurated.

Each follicle is provided with a vascular wreath which is formed by the terminal twigs of the main cortical branches.

The development of this wreath and its final obliteration, along with the disappearance of the corpus luteum, does not affect the general scheme, for it merely represents one small terminal system,

the destruction of which so far as its effect upon the general system is concerned is like the lopping off of an ultimate twig of the branch of a large tree.

For this reason the changes in the ovarian circulation incident to the progressive development and degeneration of the follicles, even in early womanhood, are local and not general. It is only in the later periods of the ovulating life of the female that the latter effect is noted. Beyond the follicular zone the terminal vessels break up into capillaries which form a fine parallel-running anastomosis in the tunica albuginea, which hitherto has not been described.

The extensive anastomosis throughout the ovary renders easy the shifting of the circulation from one set of vessels to another, consequently the abrogation of the function of the ovary is almost an impossibility before its final cessation through natural causes.

In the same way the persistence of the function in even tiny bits of the ovary, which are occasionally left after an ovariotomy, may be explained.

In order to conform to the new method of classification recently decided upon by anatomists, a system of nomenclature has been adopted by Dr. Clark which is based upon the regional distribution of the vessels.

In order to follow the progressive changes in the ovary from birth to the climateric, illustrative specimens from his collection were shown on the screen, representing the following ages: Child of two years, girls of nine and twelve years and of fourteen years, just after the establishment of ovulation; young women of twenty-four years; middle-aged woman of thirty-five years; woman approaching the menopause at forty-two years; and, finally, an old woman of sixty-six years long after the menopause.

In these specimens not only were the changes incident to the circulatory system, but also the other progressive histological transformations explained.

The comparison of this ascending series has suggested certain hypotheses concerning the physiology of the ovary which Dr. Clark believes have been strongly sustained if not entirely confirmed by the specimens in hand.

In the six months' fœtus the main branches of the ovarian artery were compared to the fasciculi of a widely-spread folding-fan, the divisions between the arteries being filled with primitive follicles.

As the ovary grows in age the vessels with the connective tissue septa which form these divisions change from a gently curved to a perpendicular course, the branches occupying the medullary portion being crowded into parallel lines (arteriæ parallelæ ovarii).

In the two-year-old child, through the development and retrogression of numerous follicles from birth up to this time, the medullary area comprises a much larger portion of the ovary than that noted in the new-born.

As there is no increase in the number of follicles after birth the obliteration of each primitive or partially developed one naturally decreases the total original number, which results in an increase in the medullary portion of the ovary at the expense of the follicle-bearing or cortical zone.

The law of development in the follicle is from within outward —that is, the primitive follicles lying nearest the central circulatory tree are the first to undergo development.

In the young child the developing follicles, instead of moving toward the periphery, as occurs in the girl approaching puberty, or in the adult, tend to maintain their primitive position, their enlargement being simply centripetal without any attempt at mobilization.

Having reached a certain stage in their development, a retrogressive change following the degeneration of the ovum is inaugurated, and the original site occupied by the follicle is replaced by a very minute addition of connective tissue to the stroma of the organ, which naturally builds up, through successive accumulations, the central area.

Follicles in various stages of development and retrogression are noted in all ages after birth, and according to Dr. Clark's observations the same principle involved in the obliteration of the unrupturred follicles before puberty governs the organization of the corpus luteum after ovulation is inaugurated.

As stated by the speaker, the changes consist in an increase in the vascular wreath around the primitive follicle and a coincident

or dependent hyperplasia of the membrana propria and an accumulation of liquor folliculi.

What determines the cessation of these progressive changes and the beginning of the retrogressive or obliterative process has not yet been explained. The fact remains, however, that with the degeneration of the ovum the liquor folliculi is absorbed and the cavity is filled in with large embryonic connective tissue cells arising from the theca interna.

Through the gradual diminution in the blood supplied by the follicular wreath the excess of connective tissue undergoes hyaline changes and absorption until finally only a mere trace of the new-growth remains.

In this way the size of the ovary is maintained within reasonable bounds. Were each mature or large follicle to be replaced by permanent connective tissue the ovary would very early in life assume the proportions of a new-growth, which sooner or later would constitute fibromata of no mean dimensions.

In tracing the progressive growth of the ovary, Dr. Clark demonstrated the obliterative changes just referred to, which continue until the follicle-bearing area, reduced by many thousands in its number of primitive follicles, becomes a narrow zone compared with its width in the new-born child.

The crowding together in more or less parallel lines of the secondary and tertiary branches of the ovarian vessels is, to return to his analogy, simulated by the partial closure of the fascieuli of the fan. According to Dr. Clark, the increase in the internal resistance through the building up of a denser medullary centre and the closer crowding together of the parallel vessels sooner or later breaks the equilibrium of forces, and consequently the follicles no longer maintain their primitive position while enlarging, but undergo mobilization toward the tunica albuginea, that being the direction of least resistance.

The actual rupture of the follicle, according to his opinion, is due to the influx of blood during the menstrual cycle into the medullary bloodvessels, which has a double action, first to push the mature follicle rapidly toward the surface, and, second, through the increased pressure, to close the parallel-running anastomosis in

the tunica albuginea, and thus favor a physiological necrosis and rupture of the follicle.

Concerning the question of ovulation and menstruation, Dr. Clark has offered further evidence to prove that the rule of synchronism is the normal, and that deviations from this rule are probably due to modifications in habits of life incident to changes in environment and to departures from primitive methods of living and from primitive laws governing sexual congress.

Brief statements were made concerning the processes through which the mature but unruptured follicles undergo obliteration. He assumed that this is not a pathological condition but is merely nature's method of getting rid of a functionless cavity. In his description of the organization of the vascular system of the corpus luteum and its retrogressive changes and final disappearance, Dr. Clark takes the position that little or nothing of the follicular vascular system remains when the resorption of the corpora fibrosa is complete.

As a conclusion to this study, the cessation of ovulation was ascribed to the gradual impairment of the vascular systems, through, first, densification of the ovarian stroma, and, second, through the retroactive effect of imperfectly removed corpora lutea, which, as an end result, diminishes the blood-supply to the cortical area to such an extent that the growth of the primitive follicles is retarded and finally completely inhibited. According to Dr. Clark, these final retrogressive changes lead up to and constitute the menopause or climacterium.

DISCUSSION.

Dr. Reynolds Wilson: It is of great interest, in view of the history of the vascularization of the ovary which Dr. Clark has given us, to know that there exists a certain class of cases of degeneration of the ovary characterized by the presence of multiple blood-cysts of varying size, due to the faulty involution of the corpora lutea. This condition is evidently dependent upon the improper organization of the blood-supply to the follicles after rupture. The degenerated ovary usually shows the presence of densely fibrous tissue surrounding the ruptured follicles, with hemorrhage

into the follicles, the blood-cyst taking the place of the normally formed corpus luteum. Such conditions are evidently due to the faulty circulation, or, rather, faulty innervation of the ovary. In these cystic conditions the blood cysts representing innumerable corpora lutea and containing a great amount of blood exert pressure upon the intervening tissue and hasten thereby the further degeneration of the ovary.

The practical point is, that these cystic conditions will give rise to symptoms simulating those of abscess formation. The resulting tumor—for such instances of degeneration may give rise to large tumors—may readily be mistaken for an inflammatory mass. I think every operator has been surprised under such conditions to find that instead of removing an abscess or suppurating mass he has found simply an enlarged ovary, or ovaries, having undergone such degeneration.

Dr. Edward P. Davis: Dr. Clark's very beautiful demonstration throws much light upon phenomena already known. Examination of the female fœtus at the seventh month has shown that the ovaries are rich in ova at this time, and that the process of ovulation is practically established. This coincides with the development of the circulation of the ovary as shown us by Dr. Clark.

It seems also probable that light may be thrown upon the production of malformations by reference to the facts in development which Dr. Clark adduces. The occurrence of hermaphroditism may receive a partial explanation by reference to the development of the embryonal circulation.

This demonstration emphasizes the clinical fact that ovulation and menstruation are dependent very largely upon the condition of the patient's blood, and that disorders of these functions cannot be properly studied without reference to the condition of the blood. Too much attention has, we think, been given to anatomical peculiarities in the pelvic organs in cases of deranged menstruation and too little to the condition of the blood.

The pain of dysmenorrhœa, in the light of this demonstration, very closely resembles the pain of headache. Both sufferings arise in organs richly endowed with blood conveyed by tortuous and finely anastomosing vessels. While anatomical lesions may account for the patient's suffering in each case, there remain a considerable number of cases in which the condition of the blood causes on the one hand headache, and equally upon the other hand pain when the phenomena of menstruation occur.

THE PLACARDING OF HOUSES FOR CONTAGIOUS DISEASE.

By ARTHUR V. MEIGS, M.D.

[Read February 7, 1900.]

At the February meeting of the College two years ago I read a paper entitled "Reasons Why the Placarding of Houses in which are Persons Suffering from Scarlet Fever and Some Other Infectious Diseases Should not be Continued." At the same time a preamble and resolution were brought forward, but their consideration was postponed in order that everyone might receive notification that the subject would be discussed. The preamble and resolution were as follows:

"WHEREAS, It is the sense of the College of Physicians of Philadelphia that the placarding of houses in which are cases of scarlet fever and some other contagious diseases, as is at present done by order of the Board of Health of this city, is more productive of harm than of good; therefore be it

"*Resolved*, That a committee of three be appointed by the President of the College to wait upon the Board of Health and represent to it the sense of this College upon the subject, and to ask that the Board cease its present practice of placarding all houses in which there occur such cases of contagious disease."

In the last two years I have had no reason to change my opinion in regard to this practice, but, on the contrary, I am more fortified in the belief that the views I held so long ago are correct. I have received letters from various sources upon this subject, and I may mention one from a distinguished physician in Chicago. The letter is dated May 23, 1898, and, after thanking me for a copy of my article, he says: "We have had in this city abundant evidence of the folly of such a practice, and it has resulted in physicians taking the

matter practically in their own hands and reporting only such cases as could be placarded without trouble."

Shortly afterward I received a letter, dated June 22, 1898, from Dr. Arthur R. Reynolds, the Commissioner of Health of Chicago, in which he writes: "I venture to send you the accompanying circulars, cards, etc., in the belief that you will be interested in knowing something more in detail of our methods of sanitary procedure by which here in Chicago we have not only overcome a pronounced hostility of the profession to the department which I found existing at the beginning of my first term as Commissioner of Health, in 1893, but have secured the cordial co-operation of the great majority of physicians in our work. I am led to send you these by a perusal of your paper read before the College of Physicians of Philadelphia—'Reasons Why the Placarding of Houses,' etc.—and recently published in the *New York Medical Record*. The keynote of my administration is to be found in the circular on 'Antitoxin Treatment of Diphtheria.'"

I have received a second letter, dated February 16, 1899, from the same physician of Chicago whose first letter to me I have already quoted. He says: "Many thanks for your letter and the accompanying reprints. I hope you will be successful in your efforts to reform your health officials. In Chicago we have a very sensible Health Commissioner. He furnishes us with postal cards for reporting contagious diseases; and if the physician states that the case is sufficiently isolated no placard is posted. The house is only placarded when the physician refuses all responsibility for isolation of the patient. This works well, pleases everybody, and gives just as much protection as the placard method."

In Chicago, upon receiving notification of the existence of contagious disease, the health authorities send a postal card to the physician in attendance, upon which the following is printed: "I hereby assume or decline the responsibility of preventing the spread of contagion in this case." The physician must strike out the word "assume" or the word "decline." Houses are placarded only if the physician declines to assume the task of preventing the spread of the contagion.

I received lately a letter from a professor in a school of medicine connected with one of the largest and oldest universities in the country. On January 26, 1900, he writes: "I thank you for your let-

ter and also for your reprint 'Placarding of Houses in Contagious Diseases.' I have read it with pleasure and agree with you perfectly. We are in the midst of the placard business in ——, and it frequently causes us to fail (?) to make a diagnosis."

I think he means by this letter that when his conscience will allow it, and his action will not bring him within the reach of the grasp of the law, he does all he can to prevent placarding. I have always done the uttermost that I have felt that it was right for me to do, and I shall do everything I can to protect patients from what I consider to be an unjust and useless intrusion upon private rights.

Some of you may remember Dr. Lainé's experience as he described it to us when this subject was discussed here two years ago. His sister's children lived in his house, and one of them was taken with a sore throat. A culture was sent to the City Hall laboratory. A day or two afterward, the child being so well that it was playing in the yard, Dr. Lainé was notified that diphtheria bacilli had been found in the culture, and that a yellow sign would be put upon his house. Dr. Lainé objected so strenuously that the official gave way for the moment, but came back a few hours later and placarded the house. Dr. Lainé went to the City Hall and protested so effectually that at the end of about two hours the health authorities sent and removed the placard. I begged him to sue the city, because I believed it was an instance in which he could have won a suit which would have been a great benefit to the medical profession, because it would have established a precedent. It seems to me that the officials placed themselves in the wrong when they put up the sign and took it down at the end of a few hours, and I think the suit could have been gained, as they acknowledged their error when they removed the placard after such a brief time.

Some time ago I was talking to a physician in the city who has a large practice, and he told me that on one occasion he was called to see a patient with diphtheria in a family in which a yellow placard upon the house would have entailed great inconvenience. Being in some doubt what to do, although quite sure the case was one of diphtheria, he sent for a tube and forwarded a culture to the city laboratory. A negative answer was returned, and he felt justified in accepting their decision, and treated the case without ever reporting it as diphtheria, although he was sure it was that disease.

Another case that I was told about was as follows: "A school-teacher was taken with a sore throat. A culture was sent to the city laboratory. The school-teacher, after a day or two, was so well that she was out riding her bicycle. At the end of two days from the time the culture had been sent to the health authorities word was sent to the school that the case was one of diphtheria. The ambulance was sent to the school to remove the teacher to the Municipal Hospital, with an order that she must go or the school would be closed. She was willing to go rather than have the school closed, but said: 'There is nothing the matter with me. Let me ride to the hospital on my bicycle.' This she was not allowed to do, but was made to go in the ambulance, in which there was another person who had true diphtheria." She was kept for a time at the Municipal Hospital for Contagious Diseases, and I have since heard that some, at least, of the medical authorities of that institution said the case never was one of clinical diphtheria, but was only bacteriological diphtheria.

I have been waiting a good while and hoping I might have the opportunity to ascertain something in regard to the presence of this diphtheria bacillus in ordinary cases of sore throat which of recent years are commonly called follicular tonsillitis, and which twenty years ago were called diphtheritic sore throat. In the Pennsylvania Hospital such attacks of sore throat are common, and especially among the servants. A few days ago one of the servants was taken with sore throat, so she worked only half a day of a Saturday. Sunday she was worse, and did no work. On Monday she was brought into the ward, and it was found that there was a good deal of whitish material over both tonsils. A culture was promptly made and was sent to our hospital laboratory. On Tuesday the white material on the tonsils was much less, and on Wednesday morning it was gone, and the patient was without fever and was nearly well. On this same day (Wednesday) a report was received from the laboratory saying that an organism had been found consistent with that of the diphtheria bacillus.

These are the cases for which placards are being put on houses by the Board of Health. This form of sore throat is one of the commonest complaints of the colder season of the year, and to call it diphtheria is too ridiculous for serious scientific men to entertain such a belief.

I attended such a case in private practice lately. The patient, a woman, had white patches upon the tonsils, and was feverish and suffered very much for one or two days. In three or four days she was well and was out-of-doors. It is in such cases as this that the officials of the city laboratory often find the diphtheria bacillus, and then they wrongfully placard the houses.

There is another matter of which I wish to speak. Last summer, according to the reports in the newspapers, a negro who had smallpox was removed to the Municipal Hospital, and with him were taken other negroes who had been living in the same house. The well persons as well as the man with smallpox were confined at the hospital until it could be ascertained if they would develop the disease. Someone interested in them sued for a writ of habeas corpus, asking that they be released from confinement, and contending that they were illegally confined. The case was tried in one of the courts, and it was decided that the health authorities were within their rights and the negroes were deprived of their liberty until released by the medical authorities. This, if it be the law, seems to me the most shocking law of which I have ever heard. It means that, if anyone of us whose wife, child, or servant should get an attack of smallpox or scarlet fever, we are entirely at the mercy of the health authorities, who can come to the house, remove every person in it, and confine them in the Municipal Hospital until the medical authorities see fit to release them.

In contrast with the way things are done in our country I want to direct your attention to the way the English manage these things. The following is from the *Lancet* of December 23, 1899:

The Peninsular and Oriental Steamship Ballaarat, which arrived at Plymouth on December 16th, reported that she had a suspected case of plague on board. The patient was one of the native crew, a coal-trimmer, about thirty-five years of age, and was taken ill two days after leaving Gibraltar, his symptoms being a high temperature and swollen glands. He was immediately isolated by the surgeon of the Ballaarat. Mr. M. F. Williams, the port sanitary officer, immediately visited the Ballaarat before anyone boarded or left the vessel, and after he had seen the patient, who is believed to be suffering from plague, proceeded to inspect the passengers and crew, Europeans as well as natives, 280 in number. Arrangements were then made for the transfer of the patient to the hospital ship Pique, in Jennycliffe Bay. The necessary purification measures were then thoroughly taken, and the Ballaarat was, at the expiration of about two hours from the time when the medical officer of health was first communicated

with, freed from detention and allowed to proceed on her voyage to London. Certain of her passengers were landed at Plymouth, but before they were permitted to do so their names, addresses, and destinations were taken down, and the sanitary authorities of each district were apprised of their approach.

Could a more perfect system be imagined? Excellent and thorough sanitary inspection and the least possible amount of detention and inconvenience caused. Contrast this with the barbarous practices of our own quarantine officials at the ports of entry into the United States.

If there were no other reason why the placarding of houses as it is at present practised in Philadelphia should stop, it would be sufficient that the practice necessarily increases very greatly the number of cases of contagious disease that are concealed. Therefore, I am fortified in the opinion I expressed two years ago, that the system is productive of a greater amount of harm than of good, and that it should be stopped.

DISCUSSION.

Dr. James Tyson: If I had known Dr. Meigs' object I would have brought with me to-night the papers from the New York Board of Health which I obtained about the time the matter was under discussion a year or more ago. I think that the sum of the law there is that only the apartment in which the infected patient is sick must be placarded, and then at the judgment of the medical inspector. Such a course seems to me entirely without objection.

In illustration of the way the law works: in the early fall I was asked to see in consultation a case which presented every feature of cerebro-spinal fever. The diagnosis was not, however, sufficiently easy to be established at a first visit. Before the second visit the patient died, but died with apoplectic symptoms. There was nothing in any way to diminish the probability of the original diagnosis. On parting with the physician he said: "Doctor, I suppose apoplexy will be a correct cause of death to place on the certificate?" I replied, "No, since the case was more like one of cerebro-spinal fever than anything else, I think it would be more nearly correct to make the certificate accordingly." "It may be," he said, "but you know that means inspection, placarding the house, and I am also liable to a fine of $100 for not recording the case." Of course, I had nothing more to say.

Thus it is that the law is being constantly evaded. Valuable statistical information, to which the Board of Health is entitled, and we, as physicians, are entitled through it, is lost to us because physicians will not take the

risk of inconveniencing their patients by reporting cases or securing bacteriological reports on them.

Dr. Daniel Longaker: I should like to add a word on this subject. I am convinced that Dr. Meigs is perfectly right in his conclusion that the practice as at present carried out is productive of far more harm than good. I had in my own family a child quite recently taken very sick with diphtheria, which, I believe, was contracted at school from a little girl whose sister was at home, sick. I am informed her illness was diphtheria, and the case was not reported. I am convinced from my own experience, and also from the experience of friends in practice in my section of the city, that the law in regard to the reporting of cases is constantly evaded. An amusing incident is that of a painter in the northern part of the city whose house was placarded because of a case of diphtheria. Everyone objected to receiving bills from him; they were perfectly willing to pay him, but he must not send any bills. Some time after this there was another case, and he successfully appealed to the doctor. This case was not reported. Every small business, it matters not what it is, is ruined and the sufferers are frequently not able to bear this. It is a question whether it is just that this burden be placed on these people. It seems to me wrong that the individual should be forced to bear the loss alone, and not the community. The law says: "The physician in whose practice a case occurs shall forthwith make the report." We know that cases of diphtheria promptly treated with antitoxin run a short course, and we can stretch the word "forthwith" and allow twenty-four or forty-eight hours to elapse. By that time the culture may show an absence of the bacillus, and thus the period of placarding is abbreviated and in my own case it was completely avoided. I went on sending my bills and no one objected, and, I may add, no harm was done.

Dr. B. A. Randall: Among the cases seen in dispensaries many are extremely suspicious, but I have found that when thoroughly cleansed by *mopping* with peroxide of hydrogen the cultures taken were invariably negative. I have often made important sanitary use of this fact, because these cases cannot be detained (perhaps many hours) until taken to the Municipal Hospital. The patient generally departs in a crowded street car, and it is a matter of extreme importance that these cases be sterilized before they leave the hospital. It has been frequently possible to put the legal culture and the subsequent responsibility in the hands of the Board of Health and at the same time make the case innocuous for a while at least. At the same time I have been able, where the letter of the law would have contravened its protective spirit, to make an immoral use of a negative report.

THE PATHOLOGY AND DIAGNOSIS OF RETRO-PERITONEAL SARCOMA.

By J. DUTTON STEELE, M.D.

[Read February 7, 1900.]

THE consideration of the pathology and diagnosis of sarcomatous tumors of the retroperitoneal space would appear to be worthy of more attention than it has received. Not only are the references in literature incomplete and usually limited to the reports of isolated cases, but the subject has been considered almost entirely from the surgical stand-point. Anything that has to do with the diagnosis of abdominal tumors should be of equal value to internal medicine, and it has been surprising to find how infrequent and inadequate are the references to the condition in the principal text-books upon the *Practice of Medicine*. The matter was brought to my attention by a tumor of this region which I had the opportunity of examining in the laboratory of the Presbyterian Hospital. As far as it can be ascertained, it is the only case of retroperitoneal endothelioma thus far reported.

The patient was under the care of Dr. De Forest Willard, to whose kindness I was indebted for the privilege of reporting the case. The specimen was shown before the Pathological Society of Philadelphia in November, 1899, and the history and pathological record were published in the proceedings of the Society.

The credit of the name "retroperitoneal sarcoma" belongs to Lobstein, and his description of the condition in his *Traité d'Anatomie Pathologique*, 1829, Tome i., p. 446, is so accurate that one can add little to it in regard to the gross appearance of the condition. He adds to his notes upon the macroscopical description that

these growths often spring from behind the mesentery, from the connective tissue sheath of the great vessels, or the fibrous tissue about the spine, but particularly from the retroperitoneal lymph-glands. He adds that the tumors are sometimes cheesy, sometimes bacony, and very often hemorrhagic. A decided capsule is usually wanting. The growth of the tumor is quick, and produces functional changes in the organs pressed upon, and often causes neuralgic pains from pressure upon the nerves.

After Lobstein, the next important observation upon the occurrence of retroperitoneal sarcoma was made by Virchow in his work *Die Geschwülste*, 1864, vol. i., p. 383. He says: "Many of the retroperitoneal tumors, known since Lobstein, are pure medullary spindle-cell sarcomata. One may doubt whether they take their origin from the fatty tissue; indeed, it is probable from their structure that they spring from the connective tissue, especially the fascias. Retroperitoneal sarcomata are, as a rule, solitary and slow of growth, but they may reach comparatively large size. The growth is influenced by surrounding pressure, and their symptoms naturally differ according to their origin. In the pelvis they first press upon the nerves, and the earlier symptoms are neuralgic pains or paralysis. In the iliac fossa the pressure is exerted upon the vessels, and the symptoms may be that of one-sided œdema or phlegmasia alba dolens. When they are higher in situation they become attached to the small gut, and they can grow so much as to entirely compress it and cause stenosis with all its symptoms."

The first and indeed the only tabulation of cases was in the article of Keresztszeghy, published in 1893. He collected nineteen cases and added two of his own. One of Keresztszeghy's cases was a lipomyxosarcoma, and as such is not included in my list, as is explained hereafter. Waldeyer's case included in Keresztszeghy's list is also an instance of a sarcomatous change in a retroperitoneal lipoma. When these two are subtracted the number of true retroperitoneal sarcomata reported in his article amounted to nineteen.

Witzel, in 1886, contributed his paper, which is considered in detail hereafter. He reports three cases, and his contributions to the diagnosis and pathology of the affection are among the most

valuable found in the publications upon the subject. The condition has been considered in short articles by Lockwood, Anderson, Vander Veer, Lizzato, and in the admirable section of Allbutt's *System of Medicine*, written by Dr. Allbutt himself. With this exception most of the references to retroperitoneal sarcoma have been reports of isolated cases, or short mention of the subject in the leading surgical text-books. The literature of internal medicine proper has been strangely silent upon the subject. Almost all the authorities consulted pass the subject by without what would seem to be due consideration.

After a somewhat exhaustive research 62 cases of retroperitoneal sarcomata have been collected. The comparative rarity of the condition is demonstrated by the fact that in 14,630 tumors collected by Gurlt, in the *Allgemeine Krankenhaus*, in Vienna, of which 894 were sarcomata, there was but one sarcoma of the retroperitoneal space. This, however, would seem too small a proportion to be a fair one. In the record of the Philadelphia Pathological Society, covering fifty-two years, there are six cases reported, including that of Dr. Willard and myself.

As has been inferred, the use of the name "retroperitoneal sarcoma," while properly applying to all growths in any structure lying behind the peritoneum, since Lobstein has been restricted to sarcomata arising behind the peritoneum, and not connected with any of the great organs in that region. This, of course, excludes those of the kidney and suprarenal capsules. Chiari's paper upon the existence of accessory suprarenal capsules in man demonstrates the fact that retroperitoneal tumors may spring from supernumerary suprarenal capsules lying in the retroperitoneal space.

Again, while tumors of the omentum and of the mesenteric glands would anatomically come under such a classification, these growths differ clinically from the so-called retroperitoneal sarcoma, and were not included among my cases. Dr. Adami, in his classical paper "Retroperitoneal and Perirenal Lipoma," calls attention to another form of sarcomatous growths. This is a lipoma or myxolipoma that has undergone sarcomatous degeneration. He cites two cases, one of Hanna and the one reported by Waldeyer, to which I have been able to add two cases reported by Vander Veer.

These growths are so nearly identical with the retroperitoneal lipomata in their clinical and pathological characteristics that they have not been classified in my list. Microscopically, fatty and myxomatous tissue predominates, and Adami's theory of their pathology appears to be the correct one—namely, that the sarcomatous development has been engrafted upon a previously existing pure lipoma or one with extensive myxomatous degeneration.

Again, only cases that are primary have been considered. There may be, of course, secondary sarcomata in the retroperitoneal glands as in other lymph-glands of the body. In such cases the primary seat is usually in the testis or ovary.

Age. The age at which the growth appears is shown by the appended table. It will be seen that the condition is most frequent in the first, fourth, and sixth decades—that is to say, with the exception of the class occurring in children, rather later than sarcoma usually presents itself. The first five years of life are quite prone to the affection, and five cases occurred in the fourth and fifth years. The years from ten to twenty-five almost entirely escape. The oldest case was a man, aged seventy-eight years, reported by Ellis; the youngest under a year of age, reported by C. A. Martin. My case of endothelioma occurred in a girl, aged four years.

0 to 10 years	.	.	10	41 to 50 years	.	.	10
11 " 20 "	.	.	0	51 " 60 "	.	.	15
21 " 30 "	.	.	7	61 " 70 "	.	.	2
31 " 40 "	.	.	9	71 " 80 "	.	.	3

Sex. Males are more frequently affected than females, though the difference is not great. In the list there were thirty-five males against twenty-three females, and in five cases the sex was not recorded.

Position. The portion of the abdomen from which the neoplasm originated is generally exceedingly hard to determine, and consequently a classification of the cases in accordance with their source is one that must be liable to error. An attempt has been made to compile such a table by assuming that tumors consisting entirely of spindle cells spring from fibrous tissue; those showing both round and spindle cells in a reticulum of fibrous tissue may have had their origin in the lymph-glands. But it cannot be said that

this classification is one of much value. The portion of the retroperitoneal space from which the growths originated are somewhat easier to determine, but even here error may creep in.

A table of the source and point of origin of the cases as far as it has been possible to determine them is as follows:

Unrecorded	7
Retroperitoneal lymph-gland of root of mesentery	8
" " " about spine	2
" " " right lumbar region	7
" " " right iliac	2
" " " left lumbar	5
" " " pelvis	2
" " " iliac region	1
" " " left lumbar and right iliac	1
Connective tissue about spine	6
" " of sheath of great vessels	3
" " of right iliac region	1
" " of left lumbar region	1
" " of left iliac region	3
" " of pelvis	1
" " about kidney	1
Doubtful origin—centre	2
" " right lumbar	5
" " right iliac	1
" " left lumbar	2
" " pelvis	1
Endothelium of lymphatic radicals about right kidney	1

It will be seen that of the 45 cases in which it is possible to arrive at even an approximate conclusion as to the tissue from which the growths spring, 28, or 64 per cent., tumors were in the retroperitoneal lymph-glands, 16, or 36 per cent., from the retroperitoneal connective tissue or the sheath of the great vessels.

The summary of the tumors in 54 cases is as follows:

Centre of the retroperitoneal space	21
Right side of " "	16
Left side of " "	11
Pelvis	4

In one case the side is not given, and one sprung from both sides. Twenty-eight, or 57 per cent., were lateral; 21, or 41 per cent., median, and 4, or 2 per cent., pelvic. The right side was oftener

affected than the left, a fact for which no explanation can be given. My case of endothelioma took its origin from lymphatics around the left kidney.

Shape and Size. The tumor is generally lobulated and usually not larger than a man's head. In its earliest stages the growth is hard and white, and it is very prone to degeneration, which is usually hemorrhagic in character. This degenerative process often goes on to such a stage that the tumor becomes cystic, and the softening may affect it to such an extent that a fluctuation wave can be felt through the abdominal wall. The tabulated tumors are cystic in 22 of the cases, or 35 per cent. In 15 of these the degeneration was a hemorrhagic one, and the centre of the softened growth was filled with dark-brown fluid or semifluid material that has occasionally been withdrawn by aspiration. One tumor is reported as cystic from mucoid degeneration. In addition eight tumors are reported as myxosarcomata. The microscopical examination in 44 cases was as follows:

Small round-cell sarcoma	9
Large round-cell sarcoma	2
Spindle-cell sarcoma	12
Spindle and round sarcoma	7
Lymphosarcoma	5
Myxosarcoma	8
Endothelioma	1

In the case reported by McGraw, the tumor, as in my own case of endothelioma, contained unstriped muscle fibres. This is easily explained by the fact that bands of unstriped muscle fibre traverse the retroperitoneal space, and the result of the inclusion of such bands by the sarcomatous process is bundles of typical unstriped muscle cells with rod-shaped nuclei that run here and there between the bundles of sarcoma cells.

Metastases occurred in 19 cases, and affected the different organs as follows:

Liver	7
Lungs	6
Spleen	2
Pericardium	2
Skin of thorax	2

Pleura, kidneys, heart, bone, brain, cord, dura—one each.

The endothelioma gave metastasis to the skin of the thorax and probably to some of the viscera.

In the greater number of cases the growth spread into the neighboring structures, following the course of the lymphatic channels. It involved the intestines in a large portion, especially in the later stages.

The tumor infiltrated and perforated the rectum in two cases, the bladder and stomach in one each. In three cases death was caused by rupture of a degeneration cyst into the peritoneal cavity.

Symptomatology. The onset of the affection is almost always insidious, and the earliest manifestations of the presence of the mass are indefinite symptoms of some interference with the function of the stomach or intestines. There may be dragging sensations in the abdomen, disturbance of digestion, constipation, diarrhœa, colicky pains, or nausea and loss of appetite.

Pressure Symptoms. The earliest symptoms that can be considered at all characteristic are those of pressure upon the venous-supply of the lower extremities, or upon the nerves of the lumbar and sacral plexuses. The manifestations of such pressure are œdema or neuralgic pains in either or both legs. These symptoms are much more characteristic of solid malignant tumors than of any other retroperitoneal growth, except, perhaps, the rare form of myofibroma, examples of which have been reported by Shields and Harris. This is probably explained by the fact that such tumors are of firmer consistency and quicker growth than any other neoplasms in the retroperitoneal space. The leg pains appear to be more frequent when the tumor springs from the lumbar or sacral region. This is easily understood when the position of the lumbar and sacral nerves is considered.

The œdema when present begins in the feet and spreads up the legs to the external genitalia and abdominal walls. Its distribution depends upon the position of the growth. In the lateral tumors the dropsy is usually unilateral, and first appears in the lower extremities of the same side, but becomes bilateral as the increasing size of the tumor causes pressure upon the cava as well as the common iliac vein. In the median variety the œdema is

commonly bilateral from the beginning. Sometimes, however, these signs of interference with the circulation and the nerve-supply are entirely absent, and the only subjective symptoms of the early stages before the presence of the tumor is observed are the signs of interference with the functions of the digestive tract. In such cases pain and œdema usually appear at a later stage of the development of the disease.

Ellis' case, a boy, aged seven years, had occasional œdema of the scrotum, left lung, and left side of the face. The tumor surrounded the left kidney. It seems possible that the pressure upon the sympathetic system caused the peculiar distribution of the dropsy in this instance.

In Keresztszeghy's case the first symptom noticed was a shooting pain in the left foot, gradually spreading over the left leg. The tumor was in the left lumbar region.

Wasting and a cachectic condition, with loss of strength and appetite, begin early in some cases, and in others appear quite slowly. Some patients, indeed, retain their strength and a fair condition of nutrition for a surprisingly long period. As the tumor enlarges the original symptoms grow worse, and new ones develop, depending upon the direction in which the pressure of the growth is exerted. These later ones are most various in character. One of the most constant is neuralgic pain felt in the lumbar region, which is often very intense, and is referred to the back of the abdomen. This and the accidental discovery of the tumor are the two most frequent causes for the patients coming under observation.

Another symptom is constipation, and sometimes intestinal obstruction, complete or incomplete. Indeed, in the middle and advanced stages of the growth the development of partial or complete intestinal obstruction is a most characteristic symptom, and special stress has been laid upon this fact by Lobstein, Virchow, and Witzel. It is due, of course, to pressure upon the small gut, or kinking from the traction of adhesions, and is usually a high obstruction in the small intestine. A case of Madelung's is quoted by Witzel, in which the occurrence of a high intestinal obstruction had thrown doubts upon a previously made diagnosis of tumor of

the genitalia. An operation showed that the growth was a retroperitoneal sarcoma.

In two cases, reported by Virchow and Elliot, femoral phlebitis —phlegmasia alba dolens—occurred. In Lurrier's interesting case blood was passed from the urethra of a child three years old, and on one occasion a piece of tissue came away through the same channel, which was shown microscopically to be from a lymph-gland. A diagnosis was made of a new growth affecting the retroperitoneal lymph-glands, and the autopsy showed a lymphosarcoma that had perforated the bladder.

In Osler's case there was polyuria amounting to 9.7 pints in twenty-four hours. The urine was of low specific gravity and without sugar. He thinks that this symptom was due to pressure upon the renal nerves and solar plexus.

Physical Signs. In the early stages of the affection the physical examination shows little besides the existence of a tumor placed far back in the abdominal cavity. In lateral growths the diagnosis between this affection and solid tumors of the kidneys and the suprarenal capsules would be extremely difficult to make by means of physical examination alone. If the mass can be distinctly separated from the liver and spleen by zones of resonance, and is movable, it is easier to distinguish a retroperitoneal growth from tumors of these organs. It must be remembered that some sarcomata spring from the right lumbar region beneath the liver, which by reason of their close attachment to the latter organ often move with respiration, and are frequently taken for solid or cystic growths of the liver itself. Such cases are reported by Ransohoff and the *Middlesex Hospital Reports*, 1888, p. 275. In E. Monnier's case the mass was closely adherent to the under surface of the liver, and projected from under its edge so that it was clearly impossible to differentiate between them by palpation.

When the tumor springs from the centre of the retroperitoneal space the examination should be made, as Witzel suggests, under deep narcosis, and after a most thorough emptying of the intestinal tract. Even then the result of such a proceedure will probably be uncertain, and the diagnosis must be made by the help of the symptoms caused by pressure upon the great vessels or the lumbar and sacral plexuses.

Position of the Colon and Small Intestine. The diagnosis is easiest when the growth has reached what may be called the middle stage of its development, since then the most characteristic symptom, namely, the relation of the colon to the mass, is most pronounced. Indeed, the sign of most importance in recognizing the retroperitoneal position of an abdominal growth is the arrangement of the intestine just referred to. It is caused by the displacement forward of the colon by the tumor as it grows between the leaflets of the mesocolon. It dissects apart the two layers of the peritoneum, and when it reaches the colon this is pushed forward, so that the bowel will lie in a groove upon the anterior surface of the tumor. If the tumor is lateral in position the ascending or descending colon will generally lie upon the anterior and inner surface of the mass.

When the tumor is median and in the upper half of the retroperitoneal space it will push up the transverse colon in the same manner.

As has been said, the relation between the tumor and the colon, and occasionally the small intestine, is most characteristic of solid retroperitoneal tumors, but it occurs in all peritoneal growths that originate near the kidneys or in the median line near the attachment of the mesentery or mesocolon, and so is not at all peculiar to sarcoma, except that solid tumors would naturally cause displacement earlier than cystic ones. Witzel has recorded the condition in two cases of tumor of the suprarenal capsule, and in one of pancreatic cyst. Shield found the transverse colon lying across the front of a myofibroma; Lockwood observed it in a case of lipoma with sarcomatous change, and Mery records a case of retroperitoneal blood cyst that carried the transverse colon forward in a similar manner.

When the tumor is lateral in position this characteristic sign appears much earlier in the course of the affection. This fact is easily explained by considering that the ascending and descending mesocolons are comparatively short and open, so that the corresponding portions of the colon are reached and pushed up so as to lie against the anterior surface of the tumor while it is still comparatively small.

As the mass enlarges, dissecting its way between the leaflets of the mesocolon, the bowel lies first on its anterior surface, and then as the tumor grows around into the flank and increases in size the colon comes to lie upon its inner as well as its anterior aspect. The presence of the bowel is usually manifested by a line of tympany which lies over the surface of the growth, with dulness everywhere else over the tumor. Splashing can also be obtained on occasions, and the tympany naturally appears and disappears, depending upon the condition and contents of the intestine. Witzel recommends that the colon be inflated through the rectum as an aid to diagnosis. In cases of lateral tumors the small intestine is usually pushed into one corner of the abdomen, generally into the upper quadrant on the opposite side of that in which the growth originated.

In the median variety, in which the tumor springs from the centre of the retroperitoneal space, directly in front or just to one side of the spinal column, Witzel claims that the colon is not pushed up by the growing forward of the neoplasm. In such cases, according to his statement, the small intestine has a somewhat characteristic arrangement. The median tumors usually are first perceived through the anterior abdominal wall in the mesogastric region, in the neighborhood of the umbilicus, and usually a little to the right of it. They dissect apart the layers of the mesentery, and the small bowel lies around their sides, closely bound to their surface. At this stage only a small portion of the tumor lies against the anterior abdominal wall, and the area of dulness is small and surrounded by a zone of tympany.

While we owe much to Witzel in return for his classification of the arrangement of the gut in the different forms of retroperitoneal tumors, still his statement that the transverse colon is not pushed forward by certain of the median retroperitoneal growths cannot be accepted as entirely correct. The transverse mesocolon is long, and a tumor growing from the retroperitoneal space, so as to dissect or spread apart its leaflets and carry forward the transverse colon, would have to obtain a considerable size before the bowel could be brought intimately in connection with its anterior surface. However, literature shows at least three cases of retroperitoneal growths

in which the transverse colon ran across the anterior surface of a median tumor.

Shield reports a case of myofibroma which filled the abdominal cavity, although it was more pronounced toward the left flank. Upon operation he found that some coils of intestine, *including the transverse colon*, lay quite flattened in the front of the tumor and adherent to its capsule. Sir Hugh Burn, in the discussion upon C. B. Lockwood's paper before the Medical Society of London, mentioned a case in which the growth was secondary to tumor of the testicle. It occupied the upper portion of the abdomen. At the autopsy there was a mass the size of a man's head attached posteriorly to the retroperitoneal fascia, and the *transverse colon* lay in front of the mass. Repeated attempts were made to demonstrate the presence of the colon during life, but unsuccessfully. It was not inflated. H. Mery reports a case of a large retroperitoneal blood cyst which sprung from a point a little to the right of the centre of the posterior abdominal wall, and occupied a good part of the right upper quadrant of the abdomen. The autopsy showed that the *dilated transverse colon ran across the tumor*, while the mesocolon furnished its anterior covering. The small intestine was pushed to one side.

Here, then, are three cases, in two of which solid retroperitoneal tumors springing from the median position, as described by Witzel, had dissected their way into the transverse mesocolon and carried the transverse colon forward, so that it lay upon the tumor's anterior surface. The third case was that of a blood cyst that, while it projected decidedly to the right, still seemed to spring from the centre of the retroperitoneal space, and had carried forward the transverse colon in a similar manner. While none of these tumors can be included in the tabulated cases, still they are retroperitoneal growths, and two at least are solid tumors identical in their clinical characteristics with primary retroperitoneal sarcoma. The conclusions to be drawn appear to be that while the transverse colon, on account of the anatomical peculiarities of its mesocolon, is not carried forward upon the surface of the retroperitoneal median growths with the same constancy as the ascending and descending colons are by lateral tumors, still the existence of such a condition in three

cases shows that its occurrence is possible, and preserves the analogy between the arrangement of the colon in both varieties of tumors springing from the retroperitoneal space. The arrangement of the small intestine, as described by Witzel, is probably more characteristic in median tumors. However, the discovery of the transverse colon upon the anterior surface of an abdominal growth would go far in determining its retroperitoneal origin.

Movability. The tumor is usually immovable, owing to its broad attachment. It can, however, show some movability. Reference has been made to the class of cases originating in the right lumbar region in which the mass moved with respiration, due probably most often to its attachment to the liver or gastro-hepatic omentum. In addition, there may be movability synchronous with respiration in growths in other positions. Stiller reports such an instance in which the tumor was situated in the upper left quadrant of the abdomen. In Witzel's case the tumor moved with deep inspiration, and sprung from around the lumbar vertebræ. R. Johnston reports a tumor that sprung from the left lumbar region which moved with inspiration. In the case reported by S. W. Morton there were two tumors, one in the lower right quadrant and one in the splenic region, that were influenced by respiration, probably on account of adhesions between them and the abdominal wall. Osler, Knapp, and the Heidelberg *Klin. Annalen* for 1840 record tumors which sprung from the median position about the spinal column, and could be moved by palpation, but were unaffected by respiration; and Pilliet and Vean record a similar case that grew from the right iliac fossa.

Course. The growth of the tumor is decidedly rapid. In thirty cases in which the duration could be definitely determined the average length of time from the beginning of the subjective symptoms to operation or death was eight and seven-tenths months. The shortest case ran thirty-two days in the case of a man reported by Chassagne. McGraw reports a man who had had indefinite symptoms for five years of slowly developing tumor in the abdomen, though the tumor grew very rapidly in the six months immediately preceding death. It seems possible that the period of rapid growth in this case represents the true duration of the malignancy of the tumor. The his-

tory, however, was so indefinite that it was considered best not to include it in the classification.

Physical Characteristics of the Tumor. The tumor is usually at first firm and hard, and is very often lobulated. Fluctuation due to cystic degeneration was quite frequently observed, but every tumor that was shown to be cystic at the autopsy did not show a fluctuation-wave during life. The explanation for this discrepancy is probably that the cystic degeneration must be so far advanced as to cause a considerable thinning of the tissue surrounding the softened centre, or that the wall must be thinned sufficiently in some part of the tumor to cause the phenomenon. In sixty-one cases nine showed well-marked and one obscure fluctuation. In the child reported by Jacoby there was a thrill felt resembling an echinococcus cyst, leading to a diagnosis of that condition. The autopsy showed, however, that the tumor consisted of a sarcoma with many cystic areas of degeneration, and Jacoby thinks that this is perhaps the explanation of the phenomenon.

Termination. The termination of the affection is usually death from exhaustion. In Stiller's case there was profuse vomiting for twenty-four hours, followed by collapse. The autopsy showed that the growth had infiltrated and perforated the stomach. In three instances death resulted from rupture of the softened tumor into the peritoneal cavity.

Diagnosis. The diagnostic features of most value, indeed, the only ones that can be considered at all characteristic, are the arrangement of the intestine on the anterior surface of the tumor; the early interference of the circulation; neuralgic pains in the legs early in the course of the affection, and later the pain in the lumbar region; the rate of growth, and the tendency of these growths to cause obstruction of the intestine.

The retroperitoneal position of the tumor should be definitely determined by the pushing forward of the colon when this sign is present, and it seems probable that a careful examination by inflation would have revealed this symptom in many more cases than in the past.

When the growth is too low in the abdomen to have any effect upon the position of the colon the interference with the circulation

is greater, and a rectal or vaginal examination will probably give data of value.

From malignant disease of the kidney the diagnosis appears to be extremely difficult. Indeed, it seems probable that it cannot be made without exploratory incision. The relations between the lumbar variety of these growths and the kidney or the suprarenal bodies are often very intimate. In almost all the lateral tumors in the list the mass was tightly adherent to the kidney or partially surrounded it. The retroperitoneal growth springing from the lower part of the abdomen, of course, could be diagnosed by its position, but in the lumbar variety and in tumors of the kidney the situation of the growths and their character are so much alike that under most conditions the diagnosis might appear to be absolutely impossible without exploratory incision. They are both round tumors, very apt to be hard or elastic, sometimes fluctuating, depending upon their condition and the amount of degeneration. The distinguishing points would be that in the earliest stages tumors of the kidney are very apt to retain their original shape, and they can be moved back into the loin more readily than tumor of the retroperitoneal space. Again, in sarcoma of the kidney, hæmaturia occurs in a good minority of cases. However, when there is no hæmaturia the urine in renal tumors usually appears perfectly normal. Indeed, other tumors of the abdomen may cause hæmaturia besides those of the kidney.

According to Rolleston and Marks, primary malignant tumors of the suprarenal capsules present a great variety of symptoms, but the clinical aspect oftenest resembles that of renal tumors. The clinical picture of Addison's disease is usually not present, but may be imperfectly suggested. Thus in two of their series of twenty-six cases there was slight pigmentation of the skin. In Palamidessi's series of thirty-eight cases of carcinoma adrenalis there were also two cases that showed pigmentation. In both series the classical symptoms of weakness and vomiting were absent. Two cases reported by Rolleston and Marks showed excessive growth of hair on the face and body. Symptoms of pressure upon the lumbar plexus occurred in one case, and œdema of the legs in six. It will be seen that the symptom-complex of retroperitoneal sarco-

mata may resemble that of tumors of the suprarenal capsule to a considerable extent, and there is no certain way of distinguishing renal, suprarenal, and lateral retroperitoneal tumors.

The distinction between retroperitoneal sarcoma and the rare retroperitoneal serous cysts may sometimes be difficult. The conclusions of Obalinski, who has made a study of such cysts, show them to be slow of growth and of tremendous size. They usually lie in the region of the kidney, and probably spring from the Wolffian and Müllerian bodies. The difference in the course, their size, and the result of puncture will be the distinguishing features.

It is perhaps unnecessary to consider aneurism of the abdominal aorta as a possible source of error. However, it is possible that such a mistake may occur, as Parker Syms reports a case of aneurism of the aorta that so closely simulated a solid tumor that an exploratory operation was undertaken.

It would be impossible, apparently, to distinguish between a myofibroma of the retroperitoneal space and a sarcoma, except by the rate of growth and resulting cachexia.

The diagnosis from lipoma and myxolipoma should be easy. The slow growth, greater size, marked fluctuation, and the late appearance of pressure symptoms in these tumors make the picture quite distinct from that of sarcoma. When, however, the lipoma begins to grow rapidly after enlarging slowly for some time a sarcomatous change should be suspected.

Tubercular enlargement of the retroperitoneal lymph-glands should usually offer no difficulty in diagnosis. The history of the case, the uniform enlargement of the abdomen with great emaciation, the common occurrence of fever, and the almost constant disturbance of the intestinal tract and diarrhœa will be the distinguishing features. The condition, moreover, is much more common than a malignant disease of the retroperitoneal glands. It is possible, however, that the nodular form of tabes mesenterica, especially that which is insidious in its growth without fever, might render the diagnosis difficult.

The fact that a mistaken diagnosis has been made in so many of the cases collected testifies to the difficulty of distinguishing this form of sarcomatous tumors from other abdominal growths. It

appears to be most difficult in the latter stages, when the growth has filled the whole abdomen, and when the relations are so disturbed that it is almost impossible to determine the starting-point of the neoplasm with any degree of certainty. The condition has been mistaken for and operated upon for hydatid cyst, ovarian cyst, tumors of the liver and spleen, and with especial frequency mistaken for malignant disease of the kidney in children.

In the present age of surgery there can be no excuse for delaying the confirmation of the diagnosis by an exploratory incision when once it has been settled that the tumor is retroperitoneal in position. The patients have everything to gain and nothing to lose by such an operation. The distinction between the so-called retroperitoneal sarcoma and other retroperitoneal growths, especially those of the suprarenal capsule and kidney, while always difficult is often impossible without opening the peritoneal cavity.

The conclusions to be drawn from the study of this series of cases may be tabulated as follows:

1. Retroperitoneal sarcomata, while rare tumors, occur with sufficient frequency to warrant more attention from the stand-point of diagnosis than they have heretofore received.

2. *Etiology.* Males appear to be somewhat more predisposed to the affection than females (56 to 24 per cent.).

The condition is more common in the first, fourth, fifth, and sixth decades of life—that is, with the exception of the first decade, much later than sarcoma usually occurs. Traumatism does not seem to play much of a part in the causation of the condition, though in three cases the connection between an abdominal injury and the appearance of the tumor seems very significant.

3. *Course.* It is a very quick-growing tumor, the average duration in thirty cases being eight and eight-tenths months.

4. *Pathology.* The tumor generally originates in the lumbar region (50 per cent.). The next most frequent seat is the centre of the posterior wall of the abdomen about the attachment of the mesentery (36 per cent.). Two per cent. grew from the pelvis. For some unexplainable reason in lateral tumors the right side

is more often affected. The tumor is usually lobulated and capsulated, and is hard and firm in its earlier stages, but is very prone to degeneration. Degenerative processes are oftenest hemorrhagic in character, but may be pruriform or myxomatous. In a third of the cases the softening progressed to such a degree that the growth assumed a cystic character.

Metastasis was not common. When it occurs the liver and lung are oftenest affected. In the majority of cases the growth involved the intestine, and the softened central area may rupture into the gastro-intestinal tract or the peritoneal cavity.

5. *Symptomatology.* The onset is insidious. The earliest manifestations of the presence of such a growth are functional disturbances in the digestive tract. The first symptoms that are at all characteristic are those of pressure upon the lumbar or sacral nerves and upon the venous supply of the lower extremities, manifested by neuralgic pains and œdema of the feet and legs.

The symptoms of the latter stages are cachexia and neuralgic pains in the lumbar region, often complete or incomplete obstruction of the small intestine, and interference with the function of various organs upon which the growth presses.

6. *Physical Signs.* In the earlier stages the physical examination is uncertain and difficult; in the middle stages the colon is pushed up and lies upon the anterior surface of the tumor. In the lateral growths the small intestine is pushed to the opposite side of the median position. This arrangement of the bowel is very characteristic of all retroperitoneal growths. The tumor may fluctuate and may move with respiration or be movable by palpation.

7. *Diagnosis.* Beyond determining the retroperitoneal nature of the growth and its malignant character, the diagnosis is very difficult. The pressure symptoms and evidences of intestinal obstruction are the most valuable differential points. It is often impossible to distinguish the growths of the retroperitoneal space from those of the kidney and suprarenal capsules by physical examination alone. The difficulty of diagnosis is, of course, increased in the latter stages of the disease when the growth fills the

abdomen, and lies in an irregular circle around those occupying the abdominal cavity more or less entirely, when it may simulate almost any of the larger abdominal tumors.

An exploratory incision is the only sure method of determining the position and character of tumors that have been shown to be situated behind the peritoneum. Surgical interference offers the only opportunity for prolonging the patient's life.

THE SURGICAL USE OF CELLULOID THREAD.

By W. W. KEEN, M.D., LL.D.,

AND

RANDLE C. ROSENBERGER, M.D.

[Read March 7, 1900.]

Seeing in the *Centralblatt für Chirurgie* for September 23, 1899, a reference to some celluloid thread recently devised by Prof. Pagenstecher, I wrote to him, asking that I might have some sent in order to test its merits. As soon as I received it I delivered it to Dr. Rosenberger, of the Jefferson Medical College Hospital Laboratory, and submit herewith the very satisfactory report made of experiments. It will be observed that both by the report and by examination of the specimens that I hand to you that the tensile strength is very great, even for very small sizes; that it is very flexible and does not easily become untied; that even without any sterilization after it has been sent from Germany in an ordinary pasteboard box, handled by myself without sterilizing my hands, and by Dr. Rosenberger without any sterilization of his hands, the thread was almost absolutely sterile (see Exp. III.).

The tensile strength is increased by almost every method of treatment for its sterilization, dry heat more than doubling it (Exp. VI.), and, what is quite as important, there is apparently no method of sterilization which is not applicable to it. It elongates slightly—*e. g.*, 1.5 kg. elongated a piece 30 cm. long to 30.5 cm. It absorbs fluids to about 41 per cent. This seems to be its only disadvantage (Exp. II.). One silk thread absorbed more, the other less, than the celluloid thread (Exp. IX.).

In my private work and at the Jefferson Medical College Hospital, Profs. Horwitz, Da Costa, Hearn and myself have used it with very great satisfaction thus far, though the use has been too recent to give a final opinion on its merits, especially as to the question of its absorbability. Dr. Rosenberger is now at work on some experiments as to this point, and we will be able before long to report the results.

Inasmuch as it has been used in a large number of operations by

Prof. Pagensteeher (*Philadelphia Medical Journal.* December 2, 1899), I have no doubt that it will prove to be a most useful material, both for suture and ligature. Compared with catgut and silk, it is much cheaper, and if it proves on extended use to be as valuable, it will go far to solve the question of the best material for sutures and ligatures.

Report by Randle C. Rosenberger, M.D.

(From the Laboratory of the Jefferson Medical College Hospital.)

The material to be examined consists of a celluloid thread, grayish-brown in color, more or less smooth, and showing upon microscopic examination to consist of interwoven fibrils with smooth edges. Measured in the dry condition, it takes size 29 by the B. and S. gauge (0.011257 inch).

Experiment I. To determine tensile strength:

Pieces of raw thread 1 metre in length supported a weight of 1630 gms. The experiment was performed as follows:

1. The thread was wrapped around a gas-fixture and around the handle of a bucket below. Water was poured into the bucket until the thread broke, when the bucket and contained water were weighed. The thread broke in nearly every instance about eight inches from the handle. 2. The thread was tied to the fixture above and to the bucket below. When tied the thread broke at the knot or in its immediate vicinity.

Experiment II. To determine absorption of fluid:

Hygrometry: Two pieces of the thread were taken, each weighing 155 mg. One piece was placed in the incubator, the other in water. The thread was dried to a constant weight in seventy-two hours, at which time it weighed 152 mg.; loss, 2 per cent. Before weighing the piece which was soaked in water it was thoroughly mopped with filter paper to rid it of excess of moisture. The thread immersed in water attained a constant weight in seventy-two hours, at which time it weighed 220 mg., a gain of 41 per cent.

Experiment III. To determine infectivity of raw thread:

As it came from the package small pieces of the thread without previous sterilization were placed in bouillon. These were incubated, and not until seventy-two hours was a growth noticed. The only organism obtained was the bacillus subtilis.

Experiment IV. To determine the effect of corrosive sublimate in aqueous solution:

The thread was soaked in a 1 per cent. aqueous solution of mercuric chloride for twenty-four hours. At the end of this time there was no

appreciable effect upon the structure of the thread, and it measured 26 by the gauge (0.01594 inch).

Influence of corrosive sublimate on tensile strength:

After soaking in a 1 per cent. aqueous solution of mercuric chloride for twenty-four hours it supported a weight of 2560 gms. (Normal, 1630 gms.; the application increased the tensile strength.) Immersed for twenty-four hours in a 1 per cent. aqueous solution of mercuric chloride, then transferred to fluid culture-media and incubated, no growth developed.

Immersion for one-half hour gave the same result.

Influence on gauge: A scarcely perceptible swelling.

Experiment V. To determine the action of corrosive sublimate in alcoholic solution:

1. Tensile strength: After soaking in a saturated alcoholic solution of mercuric chloride for twenty-four hours it supported a weight of 3120 gms. (Increases tensile strength.) Immersion in saturated alcoholic solution of corrosive sublimate for fifteen minutes rendered the thread sterile.

Influence on gauge: Slight swelling.

Experiment VI. To determine the action of formalin:

Tensile strength: After soaking in formaldehyde (40 per cent. aqueous solution for twenty-four hours it supported a weight of 2110 gms. (Tensile strength increased.) Slight swelling.

Infectivity: Immersion in formalin for one-half hour renders the thread sterile.

Experiment VII. Thermal disinfection: 1. Moist heat. *a.* At normal pressure. *b.* Autoclave at 5 K. 2. Dry heat.

a. At normal pressure (100° C.).

Tensile strength: After placing in a steam sterilizer for one hour it supported a weight of 2815 gms. (Increased.) Slight swelling.

Infectivity: Sterile after one hour's exposure.

Influence of boiling: Placed in a sterile bouillon tube and boiled for twenty minutes in a water-bath and then incubated no growth developed.

b. Autoclave at 5 K.: After placing in the autoclave for twenty minutes at 5-kilo pressure it supported a weight of 2750 gms. Great increase (50 per cent.) in strength. Slight swelling.

Infectivity: Sterile.

2. Dry heat: After placing in the hot-air sterilizer at 150° C. for twenty minutes it supported a weight of 3590 gms. (Enormous increase in tensile strength—over 100 per cent.) Slight swelling.

Infectivity: Sterile.

Experiment VIII. To determine the action of other chemicals:

a. Soaked for twenty-four hours in chloroform, there was no appreciable change except slight swelling.

b. Soaked in absolute alcohol for twenty-four hours, there was no appreciable change except slight swelling.

c. Soaked in ether for twenty-four hours, there was apparently no change except slight swelling.

d. Soaked in a 1 per cent. solution of sodium hydrate for twenty-four hours, there was no appreciable change except slight swelling.

e. Soaked in pure nitric acid, it is disintegrated in a very short time. Placed in 1 per cent. solution of nitric acid for twenty-four hours, there was no appreciable change except slight swelling.

f. Soaked in pure clove oil for twenty-four hours, there was no appreciable effect except slight swelling.

g. Soaked in equal parts of alcohol and ether for twenty-four hours, there was no appreciable effect except slight swelling.

Remarks.—The thread shows an unusual resistance to a large number of agents. It can evidently be fully disinfected in a number of ways without being in the least injured. As dry heat notably increases its tensile strength this method is to be strongly commended.

Experiment IX. Comparison with silk:

Specimen consists of two pieces of silk thread 20 cm. in length—one a plaited silk thread, the other twilled silk thread.

Upon microscopic examination with a $\frac{2}{3}$-inch objective the plaited thread is seen to consist of a number of fibrils interwoven or plaited together, but possessing, on the whole, a smooth surface. In the dried condition it weighed 210 mg., but when soaked in water it weighed 300 mg., a gain of 42.9 per cent. The gauge by the B. and S. measurement in the dried condition was 20 (0.03196 inch), but after soaking in water it measured 18 (0.0403 inch), showing that it absorbed water, which caused some swelling.

The twilled thread upon microscopic examination is seen to consist of delicate fibrils, and, on the whole, possessing a smooth surface. In the dried condition it weighed 180 mg.; when soaked in water it reached a constant weight of 220 mg., a gain of 22.2 per cent. The gauge by the B. and S. measurement in the dried condition was 16 (0.0582 inch); after soaking in water it measured 13 (0.07196 inch), showing that it absorbed a certain amount of water with considerable swelling. In both instances when the thread was weighed after soaking it was thoroughly mopped with filter-paper to rid it of excess of moisture.

THE USE OF ANTITOXIN IN DIPHTHERIA, WITH SPECIAL REFERENCE TO SMALL AND FREQUENTLY REPEATED DOSES.

By J. H. MUSSER, M.D.

[Read March 7, 1900.]

Although the value of antitoxin as a remedial measure is undisputed, many circumstances concerning its administration are debatable, among others the dosage. The writer's method of administration differs somewhat from that usually advised, and therefore he desires to engage the attention of the Fellows with the view of promoting discussion on this point. This remedial measure has been employed with the most satisfactory results, but it has not been the subject of consideration by the College. It is hoped this presentation will enable the Fellows to place their experience on record concerning this valuable antidote.

It is to be regretted a larger array of cases cannot be presented. The small number permits one only to give his impression of the effects of antitoxin. Diphtheria has not been a serious incident in the writer's practice for several years, partly because of the environment of his clientele. The cases embrace those in private and consultation practice. Dr. J. Dutton Steele was associated with me in nearly all the cases. He gave the antitoxin injections and kept the records of the cases. He carried my practice of small injections into his private work, and I am permitted to publish his cases. It gives me pleasure to make this double acknowledgment for his valuable services and kindness.

The series include instances of all ages, and most of the grades of severity of the affection, except the most severe forms in which septic complications or advanced laryngeal obstruction requiring operative measures occurred. The fact that all the cases were seen and antitoxin administered within the first twenty-four or

forty-eight hours accounts for the mildness of the disease in the series. In no instance were operative measures required, and in all recovery ensued; and, excepting in one instance of post-diphtheritic paralysis, with none of the complications so commonly seen in pre-antitoxin days.

The value of the serum treatment of diphtheria is so well established that it would be superfluous to more than refer to the fall in mortality since antitoxin first began to be universally employed. It is enough to say that a review of the literature of the subject shows that the mortality in hospital work in the pre-antitoxin period ran from 60 per cent. to 45 per cent. of all cases, and probably a slightly less percentage in private practice. The mortality of some cases treated by antitoxin varies, but shows a reduction to a very marked degree.

Laddo (*Riforma Medica*, July, 1896) records ten cases from eighty-six hospitals from all parts of the world, in which the mortality was 18 per cent. The report of the American Pediatric Society Commission, in 1896, excluding moribund cases, was 8.8 per cent. J. Herald (*British Medical Journal*, July 5, 1897) reports one hundred cases from the Kingston Ontario Hospital without a death. Baginsky (*Specielle Pathologie u. Therapie Nothnagel*), in an experience of fifteen hundred cases in hospital and private practice, compared with a similar pre-antitoxin series, stated as his opinion that the fall in mortality had been from 41 per cent. to 8 per cent. or 9 per cent. of cases treated by antitoxin. The report of the Committee of the Clinical Society of London, in 1898, gives the pre-antitoxin percentage as 29.6 per cent., and that of antitoxin treatment as 19.9 per cent. It is somewhat curious that the English percentages have not been as satisfactory as those of other countries. Perhaps this may be explained by the fact that their antitoxic serum appears to be more uncertain and weaker than the American, German, and French standards. I have been unable to determine this fact by inquiry, but Baginsky also notices that the English preparations appear to be weaker than the German.

The dosage must be necessarily fixed by empiricism. I commenced to give the serum before the publication of any statistics

of the subject could help us by the suggestion of others. From reasons of prudence the first doses given were small, and the results were so satisfactory that I have never found it desirable to increase the initial dose, and prefer to give the remedy in smaller doses and more frequently than to follow the general tendency of the time to increase the initial amount. This method implies recognition of the disease at the earliest moment possible and very close observation. The results appear to be as good as any reported, and I have had but one case of erythema and albuminuria and none of the general joint affections. The record of the cases will show that the dosage appears to have been large enough to control the course of the disease. In seven instances the temperature fell to normal and remained so in forty-eight hours. In five cases it was normal within three days, and in two very severe infections within five days.

This compares most favorably with the results of other observers. Baginsky says the "temperature should fall to normal in several days;" Monti (*Jahrbuch für Kinderheilkunde*, 1896, Band xxi., H. 1 to 3), that improvement should be noticed in twenty-four to thirty-six hours; Sidney Martin (*British Medical Journal*, 1898, ii., 624) states that in his cases eighty-three were normal before the fourth day, twenty before the eighth day, and seven after that period.

The only disadvantage we can see in the method is the pain caused by the successive injections. I have not found that this objection was sufficient to overbalance the discomfort of the urticaria and the general symptoms that appear to be more common after larger dosing. The Committee of the Clinical Society of London report that in the series of cases collected by them eruptions of some kind, apparently due to the serum injected, occurred in 37.1 per cent. of all cases, and even Baginsky himself, whose dosage appears to more closely correspond with the one here detailed, speaks of such complications as being a very frequent occurrence.

The method has been as follows: For children from naught to six or eight years the initial dose is 500 immunizing units, to be repeated at intervals of six hours if the fever does not fall, if the strength of the patient does not improve, or if the local manifestations are spreading. For children over eight years, 1000 immu-

nizing units are given as an initial dose, and this is repeated at intervals of eight to twelve hours, as needed.

Monti recommends that 1000 immunizing units be given at once in young children with moderate manifestations, to be repeated every twenty-four hours if the symptoms are aggravated or stationary. In severe cases the initial dose should be 1500 or 2000 immunizing units; if the cases are also nasal he recommends 2000 or 3000 immunizing units as an initial amount. In laryngeal cases he gives 1500 immunizing units, and repeats in twelve hours. All these doses are to be repeated within twelve hours if the symptoms do not yield.

The dose recommended by the Committee of the American Pediatric Society is as follows: For children over two years in all severe cases, and in laryngeal cases, the initial dose is to be 1500 to 2000 immunizing units, to be repeated in eighteen to twenty-four hours, with a third dose if needed; for children under two years and mild cases over two years the initial dose is 1000 immunizing units.

Baginsky's dosage is as follows: For children up to two years who are seen early in the disease, that is in the first or second day, and for mild cases 600 immunizing units are sufficient. If stenotic symptoms are present, a dose of 1000 immunizing units should be given. In older children with a mild attack of the disease the initial dose should be 1000 immunizing units. In cases of longer duration with marked glandular swelling a double dose is needed; both of these doses are to be repeated if needed within twenty-four hours. For older children, with very severe symptoms, 3000 immunizing units should be given. The second dose in all cases should be 600 to 1000 immunizing units. He adds the significant remark that one discovers very quickly that these increasing doses in severer cases, complicated as a rule with sepsis, do not accomplish very much—on the contrary, have no increased effect on the progress of the disease.

The dosage recommended by the English authorities is much larger than that given by the American and German writers. Sidney Martin and the Commission of the Clinical Society state that in their experience no initial dose under 6000 units is likely

to be effectual, and that the amount given in each case almost always reaches 20,000, and not seldom 50,000 units. As has been said, one has a strong suspicion when such doses are referred to that either the standard strength of the serum is low or that the strength is estimated on a different basis than that of other countries.

In all but one (Case I.) of the cases there was a critical fall in the temperature after the first dose of serum in the course of a few hours. In four cases the temperature fell to normal, and in the remainder the drop was at least 2° F. In Case I., which was one of the earliest cases, the initial dose of the serum was probably insufficient. The boy was twelve years old, and received a dose that I believe to be suited to a child under six years.

Baginsky refers to a transient rise of temperature after an injection of serum, first described by Variot (*La Diphtherie et la Serum Therapie*, Paris, 1898). This rise seldom exceeds 1° or 2° F., is usually not due to any complication in the disease, and may perhaps be caused, as Baginsky says, by the entrance into the system of a blood serum of a different species. It has always a short duration, and without meaning, and is followed by a fall to normal. This is well seen in Case II. and in Case XIII. after the second dose. The injection of serum has caused the rapid weak pulse, present in all the cases, to become slower and stronger in every instance.

It appears to be generally acknowledged that the serum treatment is of advantage to the heart by cutting short the process of the disease and preventing degeneration in the myocardium. As an illustration of this fact, Case VIII. is of extreme interest. A little girl who has most remarkable evidences of stenosis of the mitral and aortic valves, in whom compensation has never been completely established, but who was badly nourished and subject to attacks of dyspnœa upon the slightest exertion, came through a moderate attack of diphtheria with no signs of undue cardiac weakness.

The membrane in every instance commenced to loosen and disappear soon after the first injection of antitoxin, and its disappearance kept pace with the fall of the temperature. The toxæmia seems to lessen local resistance, and hence invite further progress

of the infection. Antitoxin is curative in the sense that the local process is thus held in abeyance. Not one case failed to show rapid local improvement. One case was treated without local applications to the throat, with very satisfactory results, and rapid disappearance of the membrane, but in the remainder of the series local remedies were used freely. Such remedies were generally hydrogen-peroxide in the proportion of one to one or one to two parts of water. In most of the cases the old-time mixture of tincture of chloride of iron and bichloride of mercury was also given. The after-treatment was interesting because we have tried most of the remedies recommended for the purpose of quickly rendering the throat free of the diphtheritic bacilli. Two were treated with a cautiously diluted spray of peroxide of hydrogen alone; two received applications of the strong nitrate of silver solution recommended by Hand. In three cases a weak solution of formalin was used as a spray. The strength of the solution was 1 to 500, and the preparation used was a proprietary one, the formula of which contained various aromatic antiseptics. One throat received no after treatment whatever. With two exceptions, the number of days required, from the disappearance of the membrane until the cultures taken from the throat were free from diphtheria bacilli, varied from four to six. We consider this variation to be so slight as to be unnecessary of notice. The throat that received no treatment was free of diphtheria bacilli in seven days, and one of the formalin cases, in which swabbing with tincture of chloride was also tried, was not free until the twentieth day.

In Case V. an erythematous rash appeared after the fifth injection. This first appeared about the point of entrance of the needle of the last dose given, and gradually involved the whole body, accompanied by a slight rise of temperature, and considerable discomfort and itching. It was of the type usually described as resembling the eruption of measles. This case also showed a certain amount of albuminuria, which was, however, transient and not accompanied by casts. In no other case was albuminuria present during the course or in the convalescence of the disease.

The serum was given in the large syringe devised for the purpose, and which can be sterilized by boiling. It is needless to say

that the injection is performed with all possible aseptic precautions, and the serum is thrown into the subcutaneous tissue of the buttock, or flank above the crest of the ilia. Care should be taken to avoid large veins.

In narrating as briefly as possible the cases, it must be borne in mind, I am constantly comparing the degree of virulence of the infection with cases of a similar character seen in the pre-antitoxic days. When I say, therefore, a case was mild, or severe, I have before me cases treated years before without antitoxin. I recall with much pain the high mortality of those days compared with the mortality of the present series. Indeed, I can say, thanks to experimental medicine, I have not seen a death from diphtheria since I have used antitoxin. I consider five of the cases were seriously ill and the prognosis doubtful had not antitoxin been used.

CASE I.—My first case impressed me very much. A lad of twelve, under the care of Dr. Murphy, of Parkesburg, for three days showed symptoms of diphtheria, and cultures were positive. On the third day at midnight I saw him. Temperature, 103.6°; pulse, 120; glands very much swollen; tonsils enlarged; fauces covered with membrane; odor characteristic; 800 immunizing units were followed by fall of temperature to 100° next morning. A second injection at 12 M. held the temperature to 101° the following evening, and the following day the temperature remained normal. The fauces improved from the first as well as the large, painful glands.

CASE II.—Dorothy K., aged three years six months. Family and previous history entirely negative. Sickened on October 20, 1897, with indefinite symptoms and some fever. Was seen by a physician, who said she had diphtheria. October 21st, seen by Dr. Musser, who found that her tonsils were covered with a diphtheritic membrane. Considerable glandular enlargement. Temperature 100.4°, pulse 140. No evidence of laryngeal obstruction. Five hundred units of antitoxin given at 9 P. M. Mother, and sister aged ten years, immunized by two hundred units. October 22d, culture shows the Klebs-Loeffler bacillus in almost pure culture. Temperature at 8 A. M. 99°. Urine: specific gravity 1016, acid, no albumin. The temperature at 6 P. M. is 100.6°. Membrane is loosening, but is somewhat blackened, and the breath is offensive. Two hundred and fifty units of healing serum given. Peroxide spray ordered every hour. Bowels opened by enema.

October 24. Morning temperature 98.4°, pulse 99. Membrane is almost gone.

25*th*. Membrane quite gone. Temperature normal all day; the patient convalesced rapidly. The cultures of the throat were negative on November 2d. Knee-jerks commenced to return. There were no complications

at any time, and no paralysis. The frequency of the pulse-rate was the most alarming feature.

Case III.—Martha E., aged four years, May 1, 1897. Had been sick for twenty-four hours with fever, sore throat. When seen there was a slight membrane on both tonsils. Temperature 99.8°. Culture taken to be examined by the Board of Health. Four hundred and fifty units of antitoxin given at 6 P. M.

May 2. Temperature 98°; membrane loosening; report of culture shows the presence of the Klebs-Loeffler bacilli; the evening temperature is 99.8°. Another dose of two hundred and fifty units of serum given, followed on the morning of May 3d by normal temperature that never again rose above normal. Considerable glandular enlargement. The antiseptic used in the throat was hydrogen peroxide, diluted one-third with water, and with the addition of a small amount of soda, to neutralize any possible amount of free acid in the preparation, every hour or two; then a proprietary preparation containing one part of formalin in five hundred of water for ten days; then a gargle, the same in a slightly stronger solution for ten days; then it was touched with tincture of chloride of iron. Culture remained positive for three weeks; no complications; no paralysis; no albuminuria.

Case IV.—Richard E., aged 12 years, May 1, 1897. Had been sick for twenty-four hours with fever and a sore throat; decided glandular enlargement; the right tonsil covered with a membrane; culture taken and sent to the Board of Health. Temperature at 6 P. M. 100°. Five hundred units of antitoxin given.

May 2. Morning temperature 100°; rose during evening to 103°. Seven hundred and fifty units of antitoxin given at 5 P. M. Culture positive.

3*d*. Temperature normal. There was no fever after this time. Antiseptics used were peroxide in the same dilution as Case II. for seven days, and the same formalin solution as in Case II. for three days. Culture negative on the tenth day. There was no albuminuria.

Case V.—Walter L., aged four years, May 11, 1897. Ill for thirty-six hours; fever, sore throat; seen by Dr. Musser 8 P.M. Both tonsils covered with membrane, which had spread to the back of the pharynx; decided obstruction to respiration, with considerable stridor; absence of knee-jerks; great glandular enlargement; temperature 102°; pulse very weak and rapid, Antitoxin five hundred units at 1 A. M.

May 13. Morning temperature 100°; membrane not spreading in the throat; considerable difficulty in respiration; free purulent discharge from the nose. Culture taken and sent to the Board of Health. Antitoxin, five hundred units, at 8 A. M. Evening temperature 100.4; general condition about the same; pulse weak and rapid; temperature at 9 P. M. 101°; urine contained a trace of albumin, but no casts or blood-cells. Antitoxin, five hundred units, at 2 P. M.

14*th*. Morning temperature 99°; membrane loosening decidedly. Five hundred units of antitoxin at 8 A.M. Condition slightly better; pulse stronger and less rapid; symptoms of laryngeal obstruction disappearing; discharge from the nose less severe; evening temperature 100°. Injected five hundred units of antitoxin at 6 P.M.

15*th*. Morning temperature 99°; antitoxin, five hundred units, at 8 A.M. Membrane loosening and disappearing in throat under the influence of a spray. Report of culture positive. A defined erythematous rash, macular in character, has commenced about the point of the last injection, and gradually spread all over the body, causing itching and some discomfort.

Morning temperature 99°, evening temperature normal. Temperature remained normal after this time. Membrane had gone on the fourth day, and the patient gradually convalesced. The eruption faded three days after its first appearance. Three weeks after the attack paralysis occurred, affecting the eyes, fauces, legs, and arms. This, however, gradually improved, and in the course of four months the child was quite well. The urine showed slight amount of albumin in the height of the attack, but this disappeared about the time of the disappearance of the fever, and did not recur. The treatment, besides antitoxin, consisted of biehloride of mercury and tincture of iron and a spray of peroxide of hydrogen with soda, diluted one-half with water every hour or two, according to the need of the case.

The case was a very severe one, and I feel confident death would have closed the scene had we not had antitoxin.

Case VI.—Elsie C., nine years of age, November 19, 1897. Had been sick with indefinite symptoms, but no complaint of the throat, for twenty-four hours. When first seen the temperature was 103°, pulse 130. Membrane on both tonsils, complete obstruction of the nasal passages, with purulent discharge from the nose. Temperature 103°, pulse 130. Knee-jerks absent; much glandular involvement. Culture taken and sent to Board of Health. Five hundred units of antitoxin given at 9 P.M.

November 20. Nine A.M., temperature 100.6°, pulse 120. The membrane is not spreading, but there is not much change in its appearance. The culture is positive. The Klebs-Loeffler bacilli are present in almost pure culture. Five hundred units of antitoxin given at 9 P.M. Temperature 101.4°, pulse 122; not much change in membrane; nose is freer, and patient is stronger.

21*st*. At 9 A.M. temperature 99°, pulse 100, and stronger; apparently is less nasal involvement. Nine P.M., temperature 99°, pulse 100, fairly strong; glands of the neck are slightly more enlarged; membrane has disappeared in the posterior wall of the pharynx. Five hundred units of antitoxin given.

22*d*. Ten A.M., temperature 99°, pulse 90; membrane disappearing rapidly. Eight P.M., temperature 99°, pulse 90; seems much brighter. At no period of the attack was there albuminuria.

23*d*. Ten A. M., temperature 98.4°, pulse 90. After this the temperature remained normal.

24*th*. The membrane has disappeared. Culture negative November 30. There was no treatment besides the antitoxin in this case. The patient was undoubtedly very ill.

CASE VII.—Mina S., aged nine years, February 14, 1898. Had had a sore throat forty-eight hours before she was seen. Membrane covers the right tonsil, and is typically diphtheroid. Culture taken, and showed the Klebs-Loeffler bacilli. Considerable enlargement of the cervical glands. Temperature at 4 P. M. 101°, pulse 140. Five hundred units of antitoxin given. Nine P. M., seven hundred and fifty units more. Temperature 101.6°.

The patient is a victim of valvular heart disease, associated with loud murmurs at the apex and in the aortic region. Murmur at the apex is presystolic, associated with a loud thrill. Murmur in the aortic region is systolic. The patient is a small, badly nourished child, with the general symptoms of insufficient cardiac compensation.

February 15. Membrane loosening about the sides. Condition of the patient is much better at 9 A.M. Throughout the day the pulse grew steadily stronger. The membrane is still loosening at the edges, and does not spread. Peroxide of hydrogen is given, diluted with three parts of water, by a spray, every two hours, during night. Temperature at 7 A. M. 100°; 6 P.M. 99.6.

16*th*. Much better; membrane is almost gone in the morning, and quite gone in the evening. Pulse is much stronger; morning temperature normal; there is no fever after this point. At no time was there urticaria or erythema, or much pain about point of injection. The urine has remained free from albumin or casts through the attack.

20*th*. Culture is negative. Case convalesced without complications; there was no paralysis. Sister, aged twelve years, has had a sore throat and nasal discharge for one week before the patient sickened. When examined, showed nothing but a large swelling of the tonsils. Baby brother immunized February 14th with two hundred units of antitoxin, and remained well. I considered the patient quite seriously ill.

CASE VIII.—Harold M., aged five years, November 5, 1897. Had an attack of laryngitis and pharyngitis, with enlargement of the bronchial tubes. In the course of three or four days he was sent to kindergarten, where he contracted a fresh cold.

November 17. White and listless. Examination revealed no membrane in morning.

18*th*. Nine A.M., first appearance of the membrane. Temperature normal, pulse 100; less prostration than on previous morning. Culture made at once, and is positive. Slight dyspnœa; symptoms of laryngeal obstruction during the day. Antitoxin, five hundred units, 8 A.M; ten A.M. there

is difficulty in breathing, with retraction of the soft parts. 1.30 P.M. all accessory muscles of respiration are in action. Antitoxin at 3 P.M., five hundred units. There seems to have been almost immediate improvement in respiratory symptoms.

19th. Culture positive. Temperature rose occasionally to 100° and 102° throughout the day. Nine A.M. received five hundred units of antitoxin—in all receiving two thousand six hundred units of antitoxin in a period of four days, in four doses of five hundred units, and three doses of two hundred units. Pulse irregular and weak during the nights of November 18th and 19th, but always came up under the antitoxin. Breathing was much embarrassed; normal in five or six days; no membrane coughed up or vomited. Culture negative in about two weeks.

CASE IX.—C. E., male, aged nine years, July 1, 1898. Had been sick with indefinite symptoms for twenty-four hours. Considerable glandular enlargement. When visited, found both tonsils covered with membrane. The clinical diagnosis was easy to make. Antitoxin, one thousand units, given at 12 M. Temperature 103.6°; at 7 P.M., 101.2°; at 10 P.M., 100°. Culture taken.

July 2d. Eight A.M. 99.4°; at 1 P.M. 100°; another dose of one thousand units antitoxin; temperature at 8 P.M. 101.4°. Membrane is loosening, and has not spread.

3d. Eight A. M. temperature is normal. Membrane disappearing rapidly. Report of culture shows presence of diphtheria bacilli. Membrane disappeared on the fourth day, and by July 9th culture was negative. The other treatment was peroxide spray of usual strength, every hour or two, and tincture of chloride of iron. No complications whatever. The patient made an uneventful recovery.

CASE X.—Arthur H., aged ten years. Seen April 24, 1899, by Dr. Musser, and the patient was found to have tonsillitis, but there was no membrane whatever.

April 26. A very small spot of yellow exudate on the right tonsil, resembling a swollen crypt. Temperature 104°.

27th. 1.30 P.M. both tonsils covered with membrane. No doubt as to diagnosis of diphtheria. One thousand units of antitoxin given. Temperature 101°; at 8 P.M. 102°. Five hundred units of antitoxin given. Considerable glandular enlargement. Culture taken.

28th. Nine A. M. temperature normal. Culture is positive, showing the diphtheria bacilli and staphyloeocci. Three P.M. temperature 101°. Antitoxin one thousand units. Urine has not shown albumin.

29th. Nine A.M. temperature 98.6°; remained normal after this. Membrane gone April 30th at noon.

When first seen there was considerable nasal involvement with a mucopurulent discharge from the nose and complete obstruction. The effect of the antitoxin at first was to loosen the membrane and to render the dis-

charge from the nose much freer. Antiseptics used were hydrogen peroxide spray in throat, the same spray somewhat diluted in the nose, and a mixture of tincture of chloride of iron and bichloride of mercury. Under the influence of the peroxide a large piece of membrane came away from the nose, giving much relief. Culture negative May 12th. Convalesced rapidly; no complications. The throat and tonsils were swabbed three times with a solution of nitrate of silver of a strength of one drachm to one ounce of water, as suggested by Hand, at intervals of forty-eight hours. Three days after the last application the culture was negative.

Case XI.—Henry D., aged twenty-six years, medical student, December 1, 1899. Has been subject all his life to attacks of tonsillitis. No previous attack of diphtheria. There was well-marked swelling of both tonsils; white membrane in upper part of right tonsil; no enlarged glands in the neck. Temperature 102°. Culture taken.

December 2. Nine A. M. temperature 100°. Membrane spreading, and black around the edges; still no glandular swelling. Six P.M. two thousand units of antitoxin given. Temperature 103°.

3*d*. Nine A. M., report of culture is diphtheria; temperature 99.6; membrane disappearing. Other treatment was the tincture of chloride of iron and peroxide. The patient was sent to the Municipal Hospital, and rapidly convalesced, with no further fever and without complications.

Case XII.—E. V., female, aged six years; April 21, 1899. Had been ill for forty-eight hours with indefinite symptoms and a sore throat. Temperature at 10 A.M. 104°. Both tonsils were covered with membrane. Slight glandular enlargement; temperature at 5 P.M. 100.6°. Antitoxin five hundred units given at 5 P.M.

April 22*d*. Seven A. M. temperature normal; membrane loosening about edges. Seven-thirty P.M. temperature 99.2°; membrane slowly disappearing.

23*d*. Temperature normal until 4 P.M., when it rose to 99°; membrane disappearing very slowly, and on April 24th, at 9.30 A.M., five hundred units of antitoxin given. Membrane disappeared so quickly as probably not to be the result of the last dose of antitoxin.

25*th*. Culture taken. Shows a few doubtful bacilli.

27*th*. Nothing but staphylococci. Convalescence rapid and without complication.

It is to be regretted that no culture was taken during the height of the illness, but the appearance of the membrane left little doubt that the clinical diagnosis was diphtheria, and the case was so treated. At no time, however, were typical bacilli found in cultures taken on blood serum. There was no albuminuria. There were two applications of the nitrate of silver solution to the throat on April 24th and 26th.

Case XIII.—B. P., aged sixteen years, who was ill from the 21st of the month. The fauces and tonsils were well covered with membrane; cul-

tures were positive; there was much odor. By the 26th the temperature was normal. The throat was not bacilli free until ten days had elapsed.

W. M., aged fourteen years, had a severe attack, and was ill three days before antitoxin was administered. The temperature rose to 102°. One thousand immunizing units of antitoxin were used, followed by prompt fall of temperature. The heart was weak and irregular. Four days after the antitoxin the child was in convalescence.

Six other cases could be reported. The case of B., seen with Dr. Anderson, was severe, and is of interest because the mother was immunized, the father was not. The latter acquired the disease.

It is not within the province of this paper to discuss the clinical varieties of diphtheria. A word may be said of securing immunity for the well by these attacks. This was attended by success in every case, notwithstanding many had been exposed. In the case of H. M., the mother and three children younger than the patient were exposed for twenty-four hours. They escaped infection, although strict quarantine could not be established.

DISCUSSION.

Dr. R. A. Cleemann: In looking over my case-book I find that I have had twenty-seven cases of diphtheria in private practice in which the diagnosis was confirmed by bacteriological test, in which I have used antitoxin. Nineteen were cases of faucial and eight of laryngeal diphtheria. Of the cases of croup two died, but died within six hours of the time antitoxin was administered; they were not intubated nor was tracheotomy performed, and I think we cannot expect the antitoxin alone to save the child when the stenosis has advanced so far. In two later cases in which intubation was performed both patients recovered. The six other cases of laryngeal diphtheria, some of them very serious, got well under antitoxin, with, in two cases, intubation, as already mentioned. All the patients were sick but a few days. The first case was practically well in three days; the second one in two days; the third one in six days; the fourth in four days; the fifth in five days, and the sixth in seven days, the last two being the intubation cases. When I say practically well I mean that the breathing was natural, the temperature normal, and there was only a little debility. They were all young children, the oldest six years. In the remaining nineteen cases, not laryngeal, there were two deaths, only one of which I think could be attributed to the diphtheria. The case of death not attributable to the diphtheria was that of a child a year and a half old which I saw in the

month of August. We were going through a heated period, and for four days the maximum of the thermometer was 90° to 96° with but little diminution of the heat in the evening. The child seemed to improve under the antitoxin as far as the throat was concerned, but on the second day I found him in a state of collapse, with a history of vomiting and purging the previous afternoon and night. The other patient who died was a very young child of sixteen months. He had been taken to the Children's Hospital suffering from fracture of the thigh. In a week's time he there developed symptoms of diphtheria and was taken home, where I gave him one thousand of the antitoxin units. The nasopharyngeal space was full of mucus, with deposit in the fauces, the child somnolent. The secretion in the pharynx became less, the membrane on one side of the throat disappeared, but the inflammation descended into the bronchi, and he died the fourth day after I saw him, with his lungs full of mucus.

My experience with the dosage is different from that of Dr. Musser. In my first cases I often used two, and in one case three, doses of one thousand units each, but in my last, about two-thirds of the whole number, as a rule, a single dose of two thousand. Using the syringe frightens the child often extremely, and I have never had bad results from giving these large doses.

In these twenty-seven cases I have seen but one with any affection of the skin. The child had a mild urticaria; very curiously, the mother had urticaria at the same time. In the few cases in which I have used the antitoxin supplied by the Board of Health the results have been as satisfactory as those obtained with antitoxin secured from other sources. Some of these cases were very severe. Among the oldest of my patients were a sister and brother, of ten and eight years respectively, taken sick at almost the same time; their fauces were full of thick membrane, their nostrils clogged, breath very fetid, and necks enormously swollen. Each had a single injection of two thousand units, and in seven and five days respectively their symptoms, even the swelling of the neck, had disappeared, and they appeared quite well. In most of the cases, however, it was a long time before the bacillus disappeared from the throat. I have used the application of sixty grains, to the ounce of water, of nitrate of silver to the throat, and have not always found that it removed the bacillus. Along with the antitoxin I have given the patients invariably chloride of iron internally, and made local applications to the throat of equal parts of tincture of iron and glycerin; but I have looked on the latter measures merely as adjuncts to the antitoxic treatment. Apart from the reduction of mortality attained by this treatment, which I think I may claim in these cases, eliminating three deaths for reasons already mentioned, as 1 in 24, or a ratio of about 4 per cent., the rapidity of cure is a most striking feature; all of the recoveries took place in from two to seven days, or an average of about four and one-third days.

Dr. F. A. Packard: In the use of antitoxin I have never seen what

some men seem to think ought to exist—reactionary fever. I think the effects are confused with tubercular antitoxin. I must say, with all due respect to Dr. Musser, that I would be afraid to use a dose of 500 units alone in any but the slightest case in the youngest child. I have never used less then 1000 units at the initial dose, and very frequently have used 2000. I think by using big doses at once we avoid the repetition. It is not very unusual to have the one primary dose sufficient to cause rapid fall of temperature, lessening of the membrane, and convalescence. I think in the end we give less antitoxin in the aggregate by giving a bigger dose in the start.

Dr. Jopson: During the past fall and winter I have seen a number of cases of laryngeal stenosis requiring intubation. I have operated on nine cases of diphtheria, in eight of which antitoxin was used. I am in the habit of recommending the administration of large doses at first, giving an initial dose of two thousand units in a patient two years of age or older, and seldom less than fifteen hundred units, even if the patient is under that age. It seems to me too much like temporizing to give the smaller doses at first in such a threatening condition as laryngeal diphtheria. Of the eight cases in which antitoxin was used six recovered and two died.

In one child, sick ten days before antitoxin was administered, the patient was almost moribund at the time I did intubation. Before I left the house the child regained consciousness, and later in the day recovered sufficiently to take food and medicines, and got out of bed when not watched. At the end of forty hours the child died of heart failure. In another case in which the antitoxin was not administered until the fifth day, the child was not only very much stenosed but very septic, and antitoxin was administered at the time the child was operated upon. Death occurred on the third day. In the remaining six the antitoxin was administered early, and all recovered.

My personal experience with antitoxin and intubation numbers twenty-three cases, including cases in which antitoxin was used very late, moribund cases, and others in which the children had bronchopneumonia at the time of operation, and many in which the surroundings and nursing were of the most unfavorable character. The mortality was a little over 50 per cent. of the entire twenty-three. Before the days of antitoxin the mortality was about 75 per cent. and recovery 25 per cent. This percentage has been about reversed, as O'Dwyer pointed out several years ago, by the use of antitoxin. I believe that in cases taken early enough the mortality will be perhaps not more than twenty.

I repeat, I am in favor of recommending in laryngeal cases the use of two thousand units in children of two years or older, as an initial dose, and seldom less than fifteen hundred units at any age.

Dr. D. J. M. Miller: I notice that none of the gentlemen have said anything about the rashes that sometimes follow the antitoxin injections. Dr. Musser spoke of one case of erythematous rash. I saw a child, of four

years, last winter, ill with rather severe symptoms of diphtheria, to whom I gave during the first twenty-four hours fifteen hundred antitoxin units. It quickly got well, but on the eleventh day it had a slight urticaria. The next day I found that the urticaria had become very extensive, and there was a temperature of 105°, with very distinct swelling of all the larger joints. I gave salicylate of soda, though the condition was probably not rheumatic. The symptoms subsided in about three or four days. Such cases must be very rare here, as but a few are reported in this country. On the other hand, European observers report many cases of urticaria and joint lesions following diphtheria.

Dr. J. A. Scott: I should like to add my voice in the interest of small doses. Within the last three or four months I have seen fourteen cases which I have treated with antitoxin. Twelve of this number were seen in one of the institutions here, a home in which we have about 110 small children. All the cases were mild, and either tonsillary or laryngeal, with one nasal case. Eleven of the children were under eight years of age. In none of these children under eight years of age did I give more than 500 units as the initial dose. In every case of the eleven but one the temperature within forty-eight hours had practically reached normal, the membrane had loosened, and eventually disappeared. In thirteen cases cultures showed the presence of Klebs-Loeffler bacillus. In one case, that of a girl of fourteen, the only symptoms manifested were tremendous enlargement of the left tonsil with great glandular enlargement. No membrane was present, but cultures showed the presence of a large number of diphtheritic bacilli. That child recovered very rapidly, the glandular swelling disappearing in a short time. In one other case there was extensive cardiac lesion, mitral and aortic disease. There was but one fatal case of the fourteen. This was a case of tonsillar diphtheria, which, on the fifth day, had symptoms of what the mother called "fainting." There was reinfection in this case, the entire tonsil and pharynx became involved, and the glands again swollen. I again used antitoxin for the fourth time, the child getting altogether 3000 or 4000 units, but on the eleventh day death occurred, without stenosis or other symptoms of laryngeal character, but of slow cardiac depression. I feel quite positively that if we can do as well with small doses of 400 or 500 units we ought to do so. Certainly, in all these children the effect was very marked.

Dr. Jay F. Schamberg: But casual mention has been made of the appearance of rashes after the use of the antitoxin of diphtheria, and the reference would make one feel, rather erroneously, that they were of uncommon occurrence. During the past few years I have been much interested in these eruptions following the employment of antitoxin, and have had the opportunity of seeing a hundred or more such cases at the Municipal Hospital for Infectious Diseases. The percentage of cases which develop rashes varies greatly at different times with the use of different brands of serum and under different observers. It frequently reaches 10 per cent. and may run as high as 30 per cent. It has been pretty well established that the

cause of the eruption is not the antitoxin itself, but the injection into the circulation of a heterogeneous blood serum. The greater frequency of rashes at the Municipal Hospital than in ordinary practice may be due to the fact that the antitoxin there employed must be used in large bulk, 10 c.c. thereof containing only one thousand units (this antitoxin is manufactured under the auspices of the Board of Health). In the large majority of cases the antitoxin eruption is urticarial; next in frequency comes the erythematous form, the patches often assuming annular and geographic outlines. In rare cases the rash is hemorrhagic, and in one case that I observed it was vesicular and bullous. At times the eruption may be scarlatiniform or morbilliform, closely simulating the rashes of scarlet fever and measles. The eruption usually makes its appearance between the first and second week following the use of the antitoxin, most commonly about the eighth or ninth day. It is frequently accompanied by a sharp rise of temperature, often 102° or 103°, and is associated with a certain amount of depression, joint pains, and at times joint swellings. I have on one or two occasions seen little ones with arms and legs tremendously swollen. The eruption usually lasts from twenty-four to thirty-six hours. With the disappearance of the rash comes a decline in the temperature and an amelioration of all of the other symptoms. The antitoxin eruptions are of much diagnostic importance because of the liability of their confusion with the ordinary exanthemata. Recently my friend Dr. Wm. Welch was called to the Broad Street Station to see a girl who was passing through our city, in whom, upon the outbreak of a rash, a diagnosis of scarlet fever had been made. He learned that the child was convalescent from diphtheria, and recognized in the rash an antitoxin eruption. These eruptions may in rare cases be delayed as long as three weeks after the injection of the antitoxin. It seems to me that the apparently current idea that antitoxin rashes are uncommon ought to be corrected.

Dr. R. A. Cleemann: I would like to add that my reason for giving large doses is illustrated in the case of a child, of four years, in which I gave at first one thousand units. This child had a patch of membrane on the right tonsil; on the third day it had disappeared and the child seemed to be well. In the afternoon of that day I was sent for and found a patch beginning on the left tonsil; I gave the child a thousand units more, and by the third day that had gone also. The thought occurred to me that had I given him two thousand units at the start the second attack would not have occurred.

Dr. Musser, closing: The reason that I give small doses is because it occurred to me that we are so frequently unable to tell whether reinfection, possibly post-nasal, may have taken place, and with it persistent toxæmia, to counteract which I thought it would be far better to give two or three doses of the antitoxin in small amounts.

I have never seen any marked reaction. I am skeptical concerning its occurrence when small doses are given.

THE DIFFERENTIAL DIAGNOSIS OF ECTOPIC GESTATION, WITH REPORT OF CASES.

By EDWARD P. DAVIS, A.M., M.D.,

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[Read March 7, 1900.]

In the classic description of ectopic gestation the patient is said to have a uterus slightly enlarged, a tumor near the womb, and the subjective signs of pregnancy. When the gestation sac ruptures, in most cases these signs become more evident because the tumor increases through the formation of an hæmatoma. These signs, taken with symptoms of anæmia and shock, point almost conclusively to the condition present.

It has been my experience to observe two cases of ruptured ectopic gestation, in which the classic signs were absent, and in which the differential diagnosis became a matter of great importance. Surgeons and physicians are called upon so frequently to treat patients in whom pain in the abdomen is associated with some degree of shock that the narration of these cases may not be without interest.

Case I. was that of a primigravida of frail physique and highly organized nervous system, the wife of a physician. This patient had been married ten years, had menstruated regularly, and had never been pregnant. She supposed herself in perfect health, and was without signs of illness when she rode upon a bicycle between one and two miles at a rapid rate, and during this ride was seized with a sharp pain in the right lower quadrant of the abdomen. She controlled her distress, however, and returned to her home upon her bicycle.

When seen soon afterward she had manifest shock; the abdomen was not swollen nor painful upon pressure, and there was no hemorrhage from

the vagina. Palpation of the abdomen was negative. The patient instinctively flexed the thighs and began very soon to recover from the shock. She was at once put at absolute rest and in charge of a good nurse. Within a few hours her husband saw her, and, at his request, a vaginal examination was made, the results of which were negative. A possible enlargement of the uterus was present, but this was scarcely noticeable. Upon questioning the patient, she remembered that she had not been unwell at the usual time, and had gone between one and two weeks over the expected date. There was no history of severe previous illness, of obstinate constipation, of mechanical injury, or of apparent hemorrhage. There was entire absence of a tumor near the womb, of a distinct enlargement of the uterus, or any evidence by examination that hemorrhage had occurred into the abdomen or pelvis. The husband was informed that ruptured ectopic gestation was possibly present. The first pain which the patient experienced occurred about 1 P. M. During the afternoon and evening the patient steadily reacted, although complaining of pain in the lower abdomen. The pulse and temperature were but slightly altered. The patient was given stimulants and morphine by hypodermatic injection. Preparations were made to open the abdomen in the early morning should indications arise.

Between 5 and 6 A.M. the patient grew worse, and as soon as possible the abdomen was opened. It was found that ectopic gestation in the right tube was present, that the wall of the tube had partially ruptured, and that the orifice had been closed temporarily by a clot. Later this had given way, and hemorrhage had taken place upward and backward among the intestinal coils. The tube was at once ligated and removed. The patient perished from shock within a few moments after the close of the operation.

In this case the diagnosis lay between ruptured ectopic gestation, disease of the appendix, and the onset of delayed and painful menstruation. This latter point may be illustrated by Case II.

Case II. was a woman who had borne two children in normal labors and who had menstruated regularly since the birth of her younger child. She was ordinarily in good health, but subject to intestinal colic and to indigestion. About the time when she should have been unwell, after exposure to cold and wet she was attacked by severe pain in the left lower quadrant of the abdomen. This pain was reflected to the right side. The entire lower abdomen was painful upon pressure, the attacks of pain were successive at intervals of from twelve to twenty hours, and on one occasion the patient was seized, while sitting, with such severe pain that it was necessary to give her an injection of morphine before she could be placed in bed. Her husband, a physician, made a vaginal examination, and finding the uterus slightly enlarged introduced a sound. The uterus was proven to be enlarged and probably empty. On examining the patient no pelvic tumor could be discovered, the womb was slightly larger than normal, the

abdomen sensitive as described, the patient's pulse slightly quickened, and her temperature normal. The attacks of pain had been so severe that the patient, a courageous woman, dreaded the possibility of return. The bowels had moved thoroughly without difficulty.

The diagnosis of ectopic gestation seemed not impossible in this case. The husband was exceedingly apprehensive that such was the condition. On close questioning, however, the history of previous attacks of intestinal colic was elicited, and it was found that the pain while severe was unaccompanied by shock. The patient was placed at absolute rest, small doses of codeia were given, the diet was limited to liquids, counter-irritation was placed over the abdomen, and the patient was carefully watched. In a short time menstruation appeared, and the patient speedily recovered.

In this case subinvolution accounted for the enlargement of the womb; the pain was that of intestinal colic, although its severity was unusual. There was but one classic symptom of ruptured ectopic gestation lacking, and that was shock. A sign of ectopic gestation so often present, a tumor in the pelvis, was absent in this case.

Ectopic gestation may be complicated by the nausea of pregnancy, and the latter may for a time obscure the diagnosis.

Case III.—Mrs. S., aged forty years, was seen in consultation with Dr. Louis Jurist in December last. Her only child was nineteen, and she had menstruated irregularly. For several months she had suffered from pain in the stomach, with loss of appetite and persistent nausea. Her menstruation had probably been absent for nearly two months. There had been a vaginal discharge of blood four weeks before she was seen, although this did not seem to be the usual period. Eight days before coming under observation she had pain in the abdomen, which subsided after she had remained in bed. The patient denied most positively the possibility that pregnancy was present. After slight improvement she left her bed and went about the house, when she was seized with sharp pain in the abdomen and severe shock. When seen by Dr. Jurist she was pulseless at the wrist, but gradually rallied.

When examined in consultation with him, the patient was pallid, restless, thirsty, her pulse 122, small and weak, the tongue dry, coated and brown in the centre, the abdomen was slightly enlarged in its lower half, painful on pressure, not tympanitic. On vaginal examination, the womb was not evidently enlarged, was freely movable with very slight pain, and there was no tumor, exudate, nor mass in the pelvis. The cervix was unaltered.

The early history of the case was of marked gastro-intestinal trouble. The patient's nausea had been irregular but persistent, chronic constipation

and dyspepsia had been present, and disease of the appendix had been strongly suggested by the symptoms. There was also a history of irregular menstruation, of previous disease of the womb or Fallopian tubes, whose exact nature could not be determined from the very obscure history given. It was my belief that the patient was suffering from ruptured ectopic gestation, the rupture occurring in the upper and posterior portions of the Fallopian tube, and the blood extravasating among the coils of intestine. She was at once transferred by ambulance to Dr. Keen's private hospital and prepared for cœliotomy. Upon consultation, two views were expressed: One, that the patient was suffering from ruptured abscess of the appendix and consequent infection of the abdomen; the other, that ruptured ectopic pregnancy was present. In view of the first opinion, I asked Dr. Keen to operate, and assisted him. An incision was made over the appendix and Fallopian tube, and extravasated blood found amid the intestinal coils. An early embryo was found just above the right cornu of the uterus, and, with a mass of blood-clot, was immediately removed. The site of the pregnancy had been the right cornu of the womb and the Fallopian tube at the point where it enters the uterus. The end of the tube was readily ligated and the torn portion removed, but persistent oozing occurred from the wall of the uterus. This was finally checked by ligating in mass and by the use of the thermocautery. Normal salt solution was poured into the abdomen and a packing of iodoform gauze carried behind the uterus, compressing the site of bleeding. This gauze was brought out at the lower end of the abdominal incision. Intravenous transfusion of fifty ounces had meanwhile been given. Although the patient's condition was critical during the operation, she reacted well. The embryo was 8 cm. long, and the chorion was found in the blood-clot removed. This patient made an uninterrupted recovery, retarded somewhat by excessive nervousness and chronic intestinal catarrh.

But little mention is made in writings upon ectopic gestation of cases in which positive evidence of pregnancy and pelvic tumor are lacking. The possibility of their absence is admitted, but held to be extremely rare. In a considerable number of cœliotomies upon women, Schauta (*Monatsschrift für Geburtshülfe*, 1900, Band xi., Heft 1) found that ectopic gestation had been present in 5 per cent. of his cases. In many of these it had not been diagnosticated, and operation was performed for pelvic tumor or tubal disease. From Case I. we observe that when the rupture in the tube is but slight it may be closed temporarily by a clot, and the patient may rally for a time from the initial rupture. An early embryo may readily die under these conditions, and, its absorption taking place,

the patient would gradually return to her accustomed health. The diagnosis of these cases may be impossible under such circumstances. When the rupture is in the right tube (and this is more frequently the case than in the left) it is very difficult to distinguish between this condition and infection of the appendix.

From these cases we believe that shock referred to the abdomen occurring in women capable of pregnancy should invariably cause a suspicion of ectopic gestation, and that, although pelvic tumor may not be found and many signs and symptoms of pregnancy be obscure, if the shock be pronounced the abdomen should be opened and the condition present accurately diagnosticated and promptly treated. We lay special stress upon the development of shock as illustrated in Case II., where its absence was the only circumstance which made the diagnosis of ectopic pregnancy, to my mind, unlikely.

A CASE OF MULTIPLE NEURO-FIBROMATA OF THE ULNAR NERVE.

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AND
WILLIAM G. SPILLER, M.D.,
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[Read April 4, 1900.]

SURGICAL REPORT BY DR. KEEN.

B. L., a laborer, from Northumberland, Pa., aged forty-seven years, first consulted me April 17, 1899.

His father died of tuberculosis; mother, of unknown cause. Neither of them suffered from rheumatism. The patient has suffered for twenty years from rheumatism, especially in his knees. For many years past there have been some tender nodules in the palm of his left hand. He is doubtful whether they are made worse by bad weather. At first they were painful only when compressed in handling a spade, axe, or any other such tool; but gradually the pain became continuous and kept him awake many nights. It was often so severe that he could scarcely refrain from outcries when it darted through his hand. As he expressed it, "The jumping toothache was not in it compared to this pain."

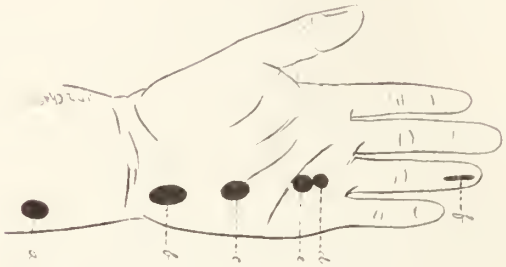
In January, 1892, another surgeon removed two of these nodules. Stellate scars at each place mark the site of the wounds, which suppurated. No microscopical examination of the tumors seems to have been made. He has now nodules at the places marked *a* to *f*, Fig. 1. These existed at the time of the prior operation, but as they were not painful they were not removed. The one in the forearm marked *a* is painful only occasionally, when, by accident, pressure is applied to it. The others are all spontaneously painful.

For twenty years also he has had a fistula in ano, for which two ineffectual operations have been done. Three openings about 3 cm. anterior to the anus, surrounded with a great deal of induration, were found.

[1] Originally prepared for the Jacobi Festival volume.

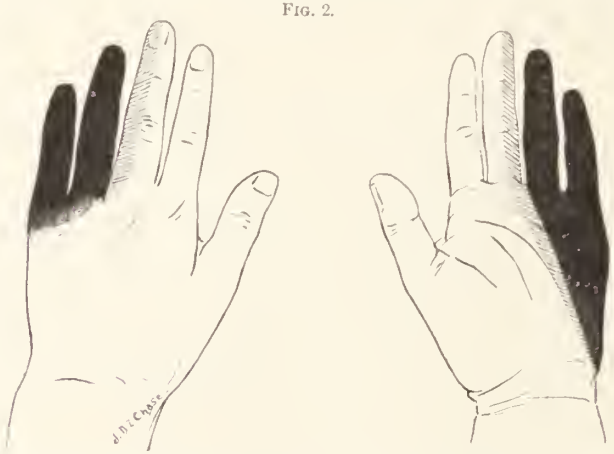
Operation, April 18, 1899. I first made an incision above the wrist and exposed the ulnar nerve (at *a*, Fig. 1), in which I found a fusiform expan-

FIG. 1.



Showing position of the tumors. Compare this with Fig. 4.

FIG. 2.



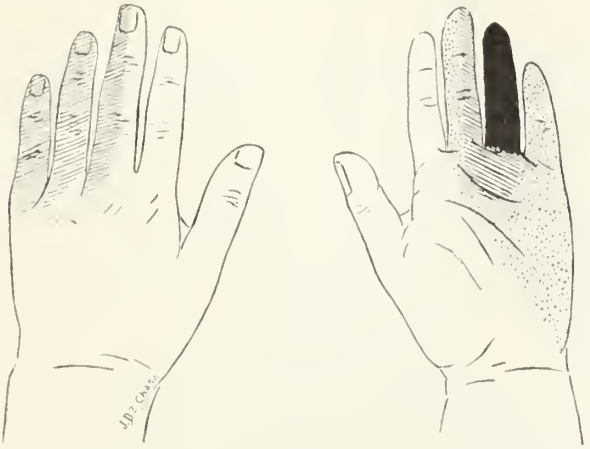
Showing the condition of sensation in the left hand of B. L., April 18, 1899, just before operation. The black area shows hyperæsthesia for all qualities, excepting cold on the median side of the ring finger. The parallel lines represent the hypæsthetic area. On the back of the middle finger the hypæsthesia was uncertain.

Sensation was tested for heat, cold, pain, and touch.

The ulnar side of the left hand was *hyper*sensitive for all qualities except on the median side of the ring finger; here cold was not perceived quite so distinctly as in the corresponding portion of the right hand. The median side of the ring finger, palmar aspect, seemed to be somewhat *hyper*sensitive to all qualities except cold. The ulnar side of the middle finger, both palmar and dorsal aspects, showed diminished sensation. When the patient was touched over the scar on the palm of the left hand—the scar made by a previous operation on the ulnar nerve—he perceived the sensation in another scar situated on the ring finger of the same hand. When he was touched over the scar in the ring finger, he located the sensation correctly. The hyperæsthesia was doubtless due to the irritation of the nerve fibres caused by the presence of the tumors. Pain prevented a firm grasp by the left hand.

sion, caused evidently by a mass in its interior. An incision made very carefully in the axis of the nerve enabled me to separate the fibres to each side, and with an Allis dissector to shell out from the interior of the nerve an oval tumor with a small filament 1 to 2 mm. in diameter at each pole of the tumor (*a*, Fig. 4). It was quite a surprise to me to find so large a tumor in view of its being so obscurely felt before operation. The same remark would hold true of all the others. A second incision was made at the eminence of the little finger (*b*, Fig. 1), and a similar tumor again shelled out

FIG. 3.



Showing the condition of the sensation in the left hand of B. L., April 27, 1899, nine days after the operation. The black area represents the nearly anæsthetic area; the parallel lines on the palmar surface represent the hypæsthetic area, and the dots the hyperæsthetic area. On the middle finger the hyperæsthesia was uncertain. On the back of the hand the parallel lines represent the slightly hyperæsthetic area.

Sensation was tested for heat, cold, pain, and touch, and the limits of the areas corresponded for each form of sensation, *i. e.*, there was no dissociation of sensation. The palmar surface of the left ring finger was almost anæsthetic in all forms of sensation. The *ulnar* side of the middle finger was hypæsthetic, but the thumb side of the middle finger and the whole of the little finger and the ulnar side of the palm as high as the wrist were slightly *hyper*æsthetic, except that the portion of the palm above the middle and ring fingers was *hyp*æsthetic; above the wrist disturbance of sensation was uncertain The thumb and first finger exhibited normal sensation.

On the dorsal aspect the middle, ring, and little fingers were somewhat more sensitive than the corresponding fingers of the right hand. The thumb and first finger were normal. The back of the hand was also normal.

of the ulnar nerve. A third incision, just above the web between the ring and the little fingers (*d*, *e*, Fig. 1), revealed two such tumors, and as I could now feel another tumor in the middle of the palm (*c*, Fig. 1), midway between the last two incisions, I made these incisions continuous, and shelled out all the tumors. On the pulp of the ring finger opposite the last

interphalangeal joint (*f*, Fig. 1), two quite small tumors were found, and the entire nerve with these two tumors was removed (see *f*, Fig. 4). All the tumors were oval in shape, with a small filament at each pole (see Fig. 4).

The fistula was then operated upon.

He made an uninterrupted recovery without febrile reaction, and left the hospital on the twelfth day entirely well from both operations.

He writes me under date of November 14th that his present condition is as follows: He has no pain, either in his hand or arm, and has full use of the hand, but still suffers from rheumatism.

Remarks. In Dr. Spiller's report on such neuro-fibromata most of the essential facts are stated, and the most important references are given and need not be recapitulated here. Operation is essential, for if left the tumor steadily increases in size. This results in constantly increasing pain, and by reason both of the pain and its consequent motor disturbances the usefulness of the hand is impaired or even lost. Moreover, from the constant pain the patient becomes very irritable. Malignant degeneration in a nerve after neuro-fibromata have been removed is by no means unknown, but the risk incurred by such an operation is justifiable in view of the symptoms produced by the tumors, and malignant degeneration may occur in these tumors even when no operation has been performed. In the present case the mode of operation was clearly indicated, and when the tumors are central in the nerve, as in this case, they should be shelled out. If they exist upon the side they should be dissected carefully from the nerve, doing as little injury to its fibres as possible, yet removing as far as may be all the diseased tissue. Whether all diseased tissue has been removed is an extremely difficult thing to determine absolutely, for from each pole of the tumor the disease may pass a considerable distance up and down the nerve, or it may exist at some other part of the nerve without causing clinical manifestations. If the tumor is too large or too intimately connected with the nerve to allow of either of these methods of operation, two resources still remain—first, a resection of the entire nerve, or amputation of the limb. The latter is not uncommonly necessary in the leg, because such tumors, in connection with the sciatic,

are apt to be very large, and also to undergo malignant change. Resection of even so large a nerve as the sciatic does not always result in total paralysis; and even should this follow, sensation and motion may occasionally be restored, either through lateral paths or by a greater or less regeneration of the nerve. Such complete regeneration I have seen personally more than once in the inferior dental nerve.

One danger attends operation, especially if all the diseased tissue has not been removed—namely, a rapid and malignant recurrence.

In Robert W. Smith's monograph, republished in 1898 by the New Sydenham Society, many admirable drawings of such tumors will be found.

PATHOLOGICAL REPORT BY DR. SPILLER.[1]

The tumor designated in Fig. 4 as *c* was studied microscopically. Sections made about half-way between the two ends of the growth showed

FIG. 4.

Actual size of the tumors removed.

[1] From the William Pepper Laboratory of Clinical Medicine, University of Pennsylvania (Phœbe A. Hearst Foundation).

that the centre of the sections was formed by dense bands of connective tissue interwoven with one another and running in various directions. Most of these fibres had a transverse course, but some ran longitudinally, and were therefore cut transversely in transverse sections of the tumor. Numerous nuclei, deeply stained by Delafield's hæmatoxylin, were found mingled with the fibres, and most of these nuclei were elongated; some, however, were round. It is difficult to determine whether the latter were

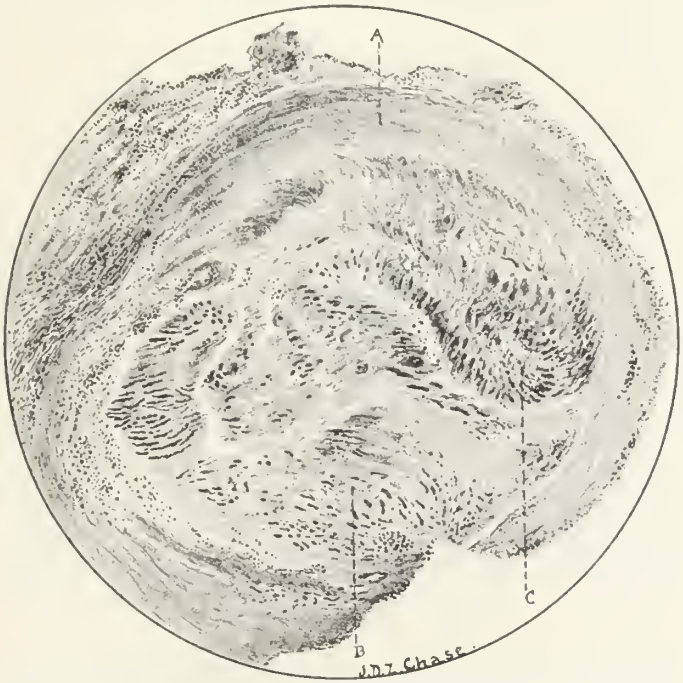
FIG. 5.

Oc. 3, Ob. 3. A section from the tumor taken near the centre of the growth. *a*, perineurium; *b*, loose connective tissue near the periphery of the growth; *c*, dense connective tissue forming the centre of the tumor.

merely elongated nuclei, cut transversely, or were really round nuclei, but probably most of them were elongated nuclei. Toward the periphery of the growth the fibrous tissue was looser and contained many nuclei, and this looser fibrous tissue passed rather abruptly into the circular bands forming the perineum (Fig. 5). Bloodvessels were rare in the centre of the tumor where the dense fibrous tissue was found, but were quite numerous in the looser tissue at the periphery. About some of the vessels at the periphery of the growth an area was found that showed a groundwork not

staining well with ammonium carmine, but containing numerous delicate wavy fibres arranged circularly about the vessel. By Weigert's hæmatoxylin method a few scattered nerve fibres were seen within the looser tissue at the periphery of the tumor, but none were found in the central dense fibrous formation. The perineurium was not much thickened. The proliferation of connective tissue evidently began in the endoneurium and in the centre of a nerve bundle. Nerve fibres were found in sections taken from

FIG. 6.



Oc. 3, Ob. 3. Transverse section from one extremity of tumor, *c*, showing the nerve fibres widely separated from one another by proliferated connective tissue. *A*, perineurium; *B C*, nerve fibres.

each end of the tumor (Figs. 6 and 7), and it was evident from these and the sections from the middle of the tumor that nerve fibres extended from one extremity of the tumor to the other, although they were not numerous.

The description of the tumor designated as *c* in Fig. 4 applies to the smaller growth designated by the letter *d* at the part marked by a horizontal line. In the tumor *d* the zone of looser fibrous tissue was between a dense central and a dense peripheral area. This small tumor also contained a few nerve fibres distinctly shown by Weigert's hæmatoxylin method. In

some of the nerve bundles removed—seen as a narrow band upon the tumor *d* in Fig. 4—and cut with the tumor *d*, the connective tissue proliferation was seen in an early stage; the individual nerve fibres were separated a little more than normally by a slight excess of connective tissue, and in some places where the connective tissue was in still greater amount the nerve fibres appeared somewhat atrophied, as though they had suffered from the pressure of the proliferated tissue. In these nerve bundles the proliferation was distinctly seen to begin in the endoneurium. These nerve bundles were distinct from the tumor *d*, but formed an integral part of the

FIG. 7.

Oc. 3, Ob. a_2 (Zeiss). Longitudinal section of tumor *c*, showing medullated nerve fibres entering at one extremity of the growth and passing to the periphery of the tumor (*A*).

tumor immediately adjoining, designated as *e* in Fig. 4. This was proof that the proliferation of the endoneurium was not confined to one or two nerve bundles within the ulnar nerve, and it would indicate that at one part of the nerve one bundle was affected, and at another part another bundle.

The piece of nerve, designated as *f* in Fig. 4, was found by microscopical sections to contain three nerve bundles presenting different degrees of thickening of the endoneurium, and the perineurium of these bundles was considerably thickened.

The connective tissue of the nerves in some persons exhibits a remarkable tendency to proliferation, and this proliferation may cause the condition known as generalized neuro-fibromatosis, in which multiple neuro-fibromata, multiple cutaneous fibromata, plexiform neuro-fibromata, elephantiasis of the skin, and pigment nævi occur.

To one unfamiliar with the literature, the classification of fibro-neuromata with some of the forms of generalized fibromatosis mentioned would seem unwarrantable, and a brief *résumé* of some of the more important papers is desirable for a proper understanding of this rather difficult subject, especially as most of the literature is in the German language.

The relation of these various pathological formations to one another has not been universally recognized. Virchow believed that a relation existed between the multiple cutaneous fibromata, the neuro-fibromata, and certain forms of congenital elephantiasis (Goldmann), and v. Recklinghausen[1] showed that the fibromata mollusca are fibromata developed on the cutaneous branches of nerves. Marie[2] states, in an excellent paper on this subject, that v. Recklinghausen's view is pretty generally accepted, but not by all. Marie does not include every fibroma molluscum under the generalized neuro-fibromatosis. In one of the two cases of generalized neuro-fibromatosis reported by him an autopsy was obtained. He was unable to find any fibromata on the nerves, or to find nerve fibres in the cutaneous tumors removed from the cadaver. The case clinically was a typical one of generalized neuro-fibromatosis, and in two cutaneous tumors removed during the life of this patient a few nerve fibres were found. The absence of nerve fibres in the tumors removed from the cadaver of course does not prove that these tumors did not have their origin in the cutaneous nerves. The cutaneous nerves are small, and may be destroyed in fibromatous thickening. The examination of the growths removed by Professor Keen has shown me that the nerve fibres within the tumor may be entirely destroyed in places by the proliferation of the connective tissue, and

[1] v. Recklinghausen. Ueber die Multiplen Fibrome der Haut, etc., 1882.
[2] Marie. Leçons de Clinique Médicale, Hotel Dieu, 1894-1895.

this is doubtless true of the nerve fibres in the fibromata of the skin.

While certain writers have contended that the multiple fibromata of the skin arise in the connective tissue of the cutaneous nerves, some make the connective tissue surrounding other structures—about the roots of the hair, glands and vessels—responsible for the proliferation. This question has not been positively decided. For example, Goldmann[1] reported a case of congenital plexiform neuroma (the first case in his paper) in which microscopically numerous fibromata were found in the skin, and all of these were in relation with cutaneous nerves. Apparent thickening of the adventitia of the vessels, of the hair sheaths, of the sweat and sebaceous glands, was in reality due to a fibromatosis of the nerves supplying and surrounding these structures. This case supports the view of the unity of the process in multiple cutaneous fibromata and multiple fibro-neuromata of the nerves. On the other hand, in a paper published by Finotti[2] three years later, different conclusions were reached. Finotti believed from his studies that the multiple fibromata did not originate exclusively in the nerves. The writers seem to agree that in structure the cutaneous fibromata and the fibro-neuromata of the nerves are very similar. The literature on this subject is given in a recent paper by Merken[3].

The generalized neuro-fibromatosis is known also as v. Recklinghausen's disease and as elephantiasis nervorum. Under the latter term Hartman[4] classes the neuro-fibromata of the nerves, the fibromata mollusca, cutaneous enlargements, and pigment spots—all are manifestations of a congenital tendency of peripheral nerves to tumor-formation. The designation of elephantiasis neuromatodes is employed by Scheven[5] to include plexiform neuromata, fibromata mollusca, and fibromatous thickening of nerve trunks.

[1] Goldmann. Beiträge zur klin. Chirurgie, vol. x. 1893.
[2] Finotti. Virchow's Archiv., vol. cxliii, 1896, p. 133.
[3] P. Merken. Wiener klin. Wochenschrift, 1899, Nos. 32, 33, and 34.
[4] Hartman. Beiträge zur klin. Chirurgie, vol. xvii. p. 177.
[5] Scheven. Ibid.

The plexiform neuroma seems to be a part of generalized neuro-fibromatosis. P. Bruns[1] has had an unusual opportunity to study this form of growth, as the study of eight cases of this rare tumor may well be considered unusual. He says that the Rankenneurome (plexiform neuroma) is one of the forms of congenital elephantiasis—known also as the fibromatous diathesis—and is the result of fibromatous thickening of the nerves of a circumscribed territory. It differs only in form from the multiple fibromata of the skin and nerve trunks. This is shown by the congenital and occasionally hereditary tendency to the formation, the simultaneous appearance of the different forms in the same person, the same histological structure, etc. Bruns was able to collect in all, including his own, forty-two cases of plexiform neuroma from the literature (1891); in three of these heredity was observed through three generations; in the first two generations multiple cutaneous fibromata, multiple fibromata of the nerve trunks, and elephantiasis occurred, and in the third generation the plexiform neuroma appeared. Bruns, therefore, classes under elephantiasis the plexiform neuromata (Rankenneurome), multiple cutaneous fibromata, multiple fibromata of the nerves, and colossal elephantiasis; and he shows that the disease may be hereditary, appearing in one form in one generation and in another in a following generation. The term elephantiasis neuromatodes seems to have originated with Bruns, but he distinguishes other forms: the elephantiasis teleangiectodes, and the elephantiasis lymphangiectodes, according as the blood or lymph-vessels are involved.

The relation between multiple fibro-neuromata and elephantiasis was recognized in two cases by Jordan.[2] The vascular system was the source of a connective tissue hyperplasia, causing great thickening of the skin and subcutaneous tissue and the formation of tumors in the nerves and muscles. Fibromata mollusca were found in both cases. In one patient the right

[1] P. Bruns. Beiträge zur klin. Chirurgie, vol. viii, p. 1.

[2] Jordan. Beiträge zur pathologischen Anatomie und zur allgemeinen Pathologie, vol. viii.

lower limb had a circumference of 75 cm. and the left 22 cm., and the right lower limb was greatly deformed. Jordan was unable to classify these cases under any of the three forms of elephantiasis (elephantiasis fibromatosa, teleangiectodes, neuromatodes) known to him. Macroscopically the condition was one of elephantiasis fibromatosa with multiple fibromata mullusca, multiple neuromata, and thickening of the large vessels; microscopically the vessels were found to be the structures in which the hyperplasia began, so that in both cases there was a combination of different forms of congenital elephantiasis. Jordan doubts whether cases of congenital elephantiasis in which only the skin and subcutaneous tissues are affected exist, *i. e.*, cases of solitary pachydermatocele.

Herczel[1] was able to observe that in a pachydermatocele the proliferation of the connective tissue originated in the thickened fibrous nerve bands; that it was a true elephantiasis neuromatodes.

The frequency of pigment nævi in generalized fibromatosis has been noted by many writers, and in a careful microscopical study Soldan[2] has recently shown that these pigment nævi (Pigmentmäler) are in the majority of cases the first recognizable signs of a fibromatous process of the connective tissue of the nerves. The different forms of neuro-fibromatosis are conditioned by the localization, anatomical relations, and energy of growth.

This brief summary of some of the most important writings on generalized neuro-fibromatosis is sufficient to show that we are justified in classing under one head the neuro-fibromata of the nerves, the cutaneous fibromata, the plexiform neuromata, certain forms of elephantiasis, and certain pigment nævi.

The causes of generalized neuro-fibromatosis are unknown. Reference has been made to the fact that heredity plays a rôle in some families, and in addition to the cases cited I may mention that Menke[3] observed neuromata in members of three

[1] Herczel. Ziegler's Beiträge, vol. viii.
[2] Soldan. Archiv für klin. Chirurgie, vol. lix., No. 2, p. 261.
[3] Menke. Berliner klin. Wochenschrift, Oct. 31, 1898, No. 44, p. 974.

generations, grandmother, mother and son, and he says a heredity through three generations existed only in the cases cited by Herczel, Bruns and Czerny. Most writers agree that the condition is not usually an acquired one. The tendency exists from the birth of the person, although the proliferation of connective tissue may not be observed until comparatively late in life. Trauma may in some cases cause the manifestation of a latent tendency in nerves.

Another important question that demands attention is in relation to the tendency of these fibromata to malignant degeneration. Several investigators have shown that this danger is not an imaginary one. Goldmann (*l. c.*) demonstrated by one of his cases that an apparently benign neuro-fibroma may undergo malignant change, or, better stated, present a malignant course, and he quotes a number of similar cases. According to him, this malignancy is not a change in the character of the tumor, but is due to the fact that a sarcoma of the nervous system may occasionally show a slow growth for a long time.

According to Finotti (*l. c.*), numerous observations have demonstrated that solitary plexiform (Herczel) and multiple neuromata (Genersick, Czerny, Winiwarter, Westphalen) have a great tendency to change into sarcomatous tissue. His words are "Umwandlung in Sarcomgewebe." Distinct clinical differences between secondary and primary sarcoma—that is, neurofibromata that have undergone sarcomatous change and those that are sarcomatous from the beginning—do not exist, according to Finotti, at least not in the majority of the cases.

Trauma may be the cause of this malignant degeneration, but in some cases no cause can be demonstrated (Garrè, Hartmann and others). Hartmann states that one of a number of neuromata may increase rapidly in size, and when it is removed by operation another tumor may develop rapidly in the same nerve trunk at the site of the former growth, more frequently, however, in another nerve territory. The second tumor is usually more malignant than the first; it involves adjoining tissue, and comparatively late metastasis occurs, causing the

death of the patient. Garrè[1] also noted the increased malignancy of the process after operation. Hartmann[2] reports a case which he says showed well the transformation of a fibroma of the nerve into a sarcoma, and he confirms the experience of others that the rapid increase in size of a neuroma, the occurrence of neuralgic pain, and sarcomatous degeneration occur at the same time. He refers also to the fact that in ten of the seventeen cases of malignant degeneration in fibro-neuromata mentioned by Garrè, death was due to a return of the tumor.

Four cases of general neuro-fibromatosis, with multiple neuro-fibromata, were observed by Thomson.[3] In the first of these one of the tumors underwent sarcomatous change, with general dissemination of sarcoma, and death after attempted removal; in the second also, after operative intervention, malignant change occurred. Scheven (*l. c.*) also refers to the pronounced tendency of the elephantiasis neuromatodes congenita to sarcomatous change, and to the fact that this tendency remains in the fibromatous nerves after a tumor that has undergone a malignant change has been removed. The surgeon can do nothing more than remove the malignant tumor; the tendency to degeneration remains in the widely-developed pathological tissue. Scheven reports also a case of malignant degeneration in elephantiasis neuromatodes, and refers to the fact that in Finotti's case the sarcomatous transformation in the neuroma could be demonstrated microscopically.

We owe to Garrè's (*l. c.*) investigations the knowledge of the frequency of the degenerative change in neuro-fibromata. Garrè was able to collect sixteen cases from the literature, seventeen with one of his own, in which sarcomatous degeneration in congenital neuromatosis had occurred. There are, of course, many more cases of malignant tumors of nerves not the result of degeneration of a fibro-neuroma. In these seventeen cases those of sarcoma arising in cutaneous fibromata are

[1] Garrè. Beiträge zur klin. Chirurgie, vol. ix. p. 465.
[2] Hartmann. Ibid., vol. xvii. p. 177.
[3] Thomson. British Medical Journal, October 10, 1896, p. 1024.

not included. Garrè showed that in an eighth of all cases of supposed benign neuro-fibromata this sarcomatous change occurs.

It is important to know what constitutes a sarcomatous degeneration in a neuro-fibroma. Rapidity of growth is suspicious according to Garrè, but is not always reliable; large size of the growth is not a positive sign, and even histologically the transformation of a benign fibroma into a sarcoma may be difficult to determine. The greater or smaller number of tumor cells is the determinative factor, but there are cases in which the diagnosis between fibroma and sarcoma cannot be made with certainty, and Garrè says that there are transitional forms between the neuro-fibroma and the sarcoma, and that these cannot be properly classed clinically or histologically. Paræsthesia, paresis, neuralgic pain, etc., are important in diagnosticating early and clinically the sarcomatous degeneration of a neuro-fibroma. The malignant change causes rapid destruction of the nerve fibres within the tumor, with the production of disturbances in motility and sensation.

It seems to me a broad and proper view to regard such neuro-fibromata as were removed by Professor Keen as an incomplete manifestation of generalized neuro-fibromatosis, although the process was confined to one nerve, the ulnar. The fibromatosis does not differ from that occurring in cases with more extensive clinical manifestations, and the limitation of the process so far is no proof that later we shall be unable to trace evidences of a more general fibromatous change. A number of cases have shown that the fibromatosis of nerves may remain undetected until the patient is well advanced in years. We understand likewise that Professor Keen's patient is exposed to the danger of malignant growth at any time.

The location of the tumors in Professor Keen's patient is especially interesting. Garrè, in speaking of his case, says that the presence of multiple small fibromata in the skin of the sole of the foot was very remarkable, and refers to the fact that v. Recklinghausen emphasized the immunity of this part and of the palm of the hand. Marie (*l. c.*) likewise says that

in generalized neuro-fibromatosis the tumors are not usually found in the hands and feet.

It is curious that only certain nerve bundles of the ulnar nerve were affected in Professor Keen's patient, but such a condition is well known. Goldmann (*l. c.*) says it is difficult to understand why in the same nerve trunk, even in the same bundle, the proliferation may involve only certain groups of fibres.

Bowlby[1] describes a specimen of multiple fibromata on a single nerve (posterior tibial) seen in the museum of the Middlesex Hospital. No tumors were found on any other nerve. This seems to have been the only case of the kind which had come under his observation when he wrote his book. A similar case was published by J. K. Mitchell,[2] and W. J. Taylor operated on a patient with neuro-fibromata of the foot, probably confined to a single nerve. As a contrast to Professor Keen's case and to Bowlby's, I may mention that Smith reported one case in which the total number of neuro-fibromata existing upon the nerves removed from the body exceeded 800, and another in which upward of 1400 neuromatous tumors were removed with the nerves, and he felt that he was not exaggerating in stating that this patient must have had at least 2000 tumors. This work of Smith was originally published in the form of a very limited edition fifty years ago, but it was deemed of sufficient importance to be reprinted in 1898 by the New Sydenham Society. Some of the plates present nerves covered thickly with fibro-neuromata, but no neuromata shelled out of a nerve, as in Professor Keen's case, are pictured in this atlas. Indeed, Smith[3] stated that the results had not afforded much encouragement to the practice of dissecting out the tumor from the branches of the nerve among which it was entangled. According to Smith, there are few affections more rare than neuro-fibroma.

[1] Bowlby. Injuries and Diseases of Nerves and their Surgical Treatment, p. 493.

[2] J. K. Mitchell. The University Medical Magazine, November, 1897.

[3] Smith. A Treatise on the Pathology, Diagnosis, and Treatment of Neuroma. The New Sydenham Society, 1898.

Smith was not able to trace nerve fibres through a neuro-fibroma, although in a few instances he observed some nervous filaments entering the superior extremity of the tumor. I have been able to detect the presence of nerve fibres in both extremities and also in the centre of one of the tumors (*c*, Fig. 4) removed by Professor Keen, and my success was probably due to the fact that I had Weigert's hæmatoxylin stain at my command. In the smaller tumor (*d*) nerve fibres were also seen within the tumor. This method was unknown when Smith wrote his treatise.

It is a merciful provision that the multiple neuro-fibromata are usually painless, and it seems extraordinary in contrast that the solitary tumor is often painful.

Many investigators have observed that the proliferation begins in the endoneurium, as it did in Professor Keen's case. It is due to this mode of origin that these neuro-fibromata are elongated with their long axes parallel to the nerve. The perineurium offers a certain amount of resistance, especially as it often becomes thickened simultaneously with the growth of the tumor, and the fibroma grows especially in the direction of least resistance. In the tumor designated as *c* in the drawing (Fig. 4), the perineurium formed a sheath, surrounding on all sides the proliferated endoneurium, and in the centre of the tumor no nerve fibres could be detected, except at the periphery. This proliferation of the endoneurium was evident also in the tumor *d* (Fig. 4), and in small nerve bundles, adjoining the tumor *d* and removed with it, the proliferation of the endoneurium could be detected in its early stages. Neuro-fibromata do not always originate in the endoneurium. In a case reported by Finotti, for example, the fibroma began in the epineurium and was adherent to the nerve.

The entire nerve on which the neuroma is formed may lie in the centre of the tumor or surround the tumor as a sheath. According to L. Bruns, the nerve fibres caught within the neuro-fibroma show a remarkable resistance to degenerative

[1] L. Bruns. Die Geschwülste des Nervensystems.

processes, which explains the absence or mildness of the clinical symptoms in many cases. I am unable to fully confirm this statement from the examination of the tumors removed by Professor Keen. In two tumors, *c* and *d*, I found the nerve fibres within the tumor entirely destroyed, except at the periphery of the tumors, while in the piece of nerve marked *f*, in which the proliferation was not excessive, the nerve fibres were quite well preserved. I would prefer to explain the absence or mildness of the clinical symptoms by the slowness of the process, by the fact that only here and there a nerve bundle is attacked, and that many fibres—the majority, in fact—remain intact and do not lose their function, although considerably compressed, as the process is slow enough to allow the nerve fibres to become accustomed to the pressure.

THE MANUSCRIPT LETTERS OF JENNER IN POSSESSION OF THE COLLEGE.

BY S. WEIR MITCHELL, M.D.

[Presented April 4, 1900.]

I HAVE looked with care over the various lives of Jenner and the histories of vaccination, and fail to find any of the letters which are in possession of the College. The letters are characteristic and show that Jenner was a very quick-tempered man. He had no charity as to what he termed the barbarous murders committed by the unchecked smallpox. These letters, seventeen in number, were written to Charles Murray, Esq., 17 Bedford Row, Bedford Street, London. They begin in 1806, and the last bears the date of June, 1817.

Charles Murray was secretary to one of the societies which had for their object the propagation of Jenner's views and practice.

Of the Dr. Walker, who is spoken of so angrily, we get but an imperfect account in Baron's *Life of Jenner*. This biographer wrote too soon after Jenner's death, and deals rather tenderly with many who were still alive. Hence we get no distinct idea of what were the errors of Dr. Walker which aroused Jenner's wrath, nor is it more clear as to what gave rise to a still more positive demonstration of dislike in regard to the Mr. Adams whom he calls "the apostate." In Baron's life he is described as having made certain experiments on smallpox, and in his own opinion had produced a very mild and innocuous form of the malady, presumably to be used for inoculation. Baron, as usual, tells little of inter-

est as to this, and perhaps in the range of biographical literature there is no life of a man of genius more disappointing than this record of Jenner.

Dr. Walker had been appointed resident inoculator and Medical Secretary of the Royal Jennerian Institution, of which Jenner was president. Dr. Jenner's charges finally caused Dr. Walker to resign in March, 1806.

The letters of Jenner in possession of the Philadelphia College of Physicians were bought in England and presented to the college by Professor Da Costa and Dr. S. Weir Mitchell. They are certainly genuine, and such of them as throw light on Jenner's character I herewith submit for publication. The letters which I do not include are letters on business or mere friendly notes. They seem to show a close intimacy between the great physician and his correspondent.

I possess also an interesting letter of advice from Jenner to Mr. Monroe, then United States Minister to the Court of St. James. It has not been printed, and I add it to these letters because of its sagacious statement as to the epidemic influenza, from which Mrs. Monroe had suffered.

[Jenner Letter, No. 1.]

MY DEAR SIR: It seems to be the order of the day I think for the very men who make laws to break them. The late disorderly proceedings of our Society must be vexatious to every well wisher to its interests. It is my intention to be in town as soon as I can with any degree of convenience steal away from the country, and I shall come with the hope of arranging some plans for placing the Society on a better footing than it stands at present. Considering the magnitude of its object and (I flatter myself) the credit of the discovery which gave it birth to the British Nation, I cannot but look upon it as an object worthy the patronage of the Legislature. My only fear is a temporary suspension of the design from the vast importance of those state affairs which now so fully occupy the minds of the Ministry.

I wrote to Mr. Angerstein almost immediately after you communicated to me the unfortunate intelligence respecting my friend Travers. As I have not yet seen his name in a certain list I shall indulge the hope that his affairs may take a favorable turn.

My sentiments respecting the general conduct of Dr. Walker are now, I imagine, very generally known to the Board of Directors and the Medical Council, as I expressed them without reserve to some of the leading mem-

bers of both Committees. I now feel fully convinced that neither the persuasion of friends nor the menaces of enemies will ever produce in Dr. Walker that correct line of conduct which the Society has a right to expect in the Medical Secretary and resident Inoculator. With the hope of seeing you in town I remain Yours very faithfully,

E. JENNER.

BERKELEY, 12 April, 1806.

To CHARLES MURRAY, ESQ., London.

[Jenner Letter, No. 3, to Charles Murray.]

MY DEAR SIR: Previously to my receiving your packet directed to me at Cheltenham I consigned a letter for you to the care of Mr. Paytherus, which I hope has reached you.

If you had not acted as you did on receiving so gross an insult from the apostate Adams I should say the laws of attraction were not immutable. Your fingers were compelled like the needle to the magnet to fly to the centre of his countenance after the excitement they received. This is not the only chastisement this gentleman will receive. You have very properly inflicted corporal punishment on the delinquent and if I mistake not the world will give him enough of the mental. I do not consider Dr. Adams as any longer a fit man to conduct the business of vaccination at the smallpox hospital, as his late writings on the subject evince a spirit of disaffection. How can such a man be trusted?

I am much obliged to you for sending me Dr. McKenzies Report, which shall be returned to you on my coming to town. We can make some striking extracts from it for the papers and the medical journals; and which will serve also for the benevolent purpose of my friend Mr. Bernard. From the torpor into which the B. I. soc. has fallen, it will be most prudent I imagine to give up all thoughts of a festival this Season. We shall have a fair excuse in the new arrangement about to be made by Government. Of what nature this will be, whether the giving strength to the present Society by a pecuniary grant or building up a new system for facilitating the progress of vaccination I know not. If called upon I feel myself at a loss what to advise. Should any good plan occur to you dont fail to commit it to paper that I may see it when we meet. I mention something on the subject in a letter lately to Mr. R. Phillips and wish you to look at it.

Believe me, Yours truly, E. JENNER.

April 29, 1808.

[Jenner Letter, No. 4, to Charles Murray.]

MY DEAR SIR: If Mr. Bliss, who has always been represented to me as a sensible man, considers the case[1] in Mr. Gambier's family as a failure how

[1] This case is not mentioned in Baron.—ED.

miserably have I been wasting my time and labor in making experiments to ascertain the laws and agencies of the variolous and vaccine virus on the human constitution and in endeavoring to impress those laws on the minds of the faculty. This child, it seems, was found unsusceptible of the effluvia of the variolous poison, but not of the venom when inserted into the skin. Where then is the failure of vaccination? Was it not proved by the natural test that the child in question had actually received security from the contagion of the smallpox? And even when inoculated the consequences were exactly such as have happened to those who have previously had the smallpox in a vast number of well attested instances. After this I may exclaim with Cobbett, "It is done." At least I think it high time that I should have done with it.

I have seen Dr. Lucas Pepys this morning. He thinks that no time should be lost in Dr. Rose's being set right with regard to his mistatements in the House of Commons by showing him the reports, etc. Will you be good enough to refer Mr. Bliss to what I have said on the subject which immediately relates to the case of Mr. Gambier's child? The reference I mean is to my book, which Mr. B. probably has in his possession.

Very respectfully yours, EDWARD JENNER.

June 23, 1808.

[Jenner Letter, No. 6, to Charles Murray.]

MY DEAR SIR: This second messenger that you have sent after a letter there is no resisting. Pray tell Mr. Walker that my coming to town this spring is very uncertain, and that I am fearful that it may not be in my power to pay my annual visit to his nursery; and be so good as to say that I think the vaccine lancet just as safe in the hands of Mr. Ring as in my own. To go back to your first letter. You argue the point ably and judiciously respecting the last piece of intelligence I communicated to you concerning the Establishment. That crowns the whole. It cannot be a secret. An experimental inquiry made by a public Body and acting under the eye of Government can never be conducted in the dark; therefore speak of it as much as you like, but say nothing either of Mr. B. or myself when you mention it. It will be most prudent I think for me to remain quiet and not to notice the transactions of the Institution till called upon from some respectable quarter, and that I think will be the case. In these days of keen investigation I should not be surprised to find a question put to the Minister as to the expenditure of 3000 per annum. When any public notice is taken of the affair, then, and not till then, I conceive will be the time for me to come forward. As for the calumnious whispers that go about town I regard them not a rush. Having according to the best of my judgement acted conscientiously in the matter my mind is quite at ease, and I trust will be so, follow what may. I strongly surmise that this extraordinary plan of investigation originated in the prejudices imbibed by a

certain gentleman from the blunders committed by the vaccinators at Ringwood, which you know produced disasters. But this, inter nos. Why not institute an enquiry into the validity of the magnet because a heedless mariner sometimes runs his ship upon a rock?

In your letter of the 10th I see you suppose that I had not written to Mr. R. You were wrong in your conjecture. I wrote very fully to him, but he chose to misunderstand me, because perhaps he is unaccustomed to hear a plain unvarnished tale. He still harped upon the unreasonableness of my wishing to take the whole management into my own hands, though I assured him nothing was more distant from my thoughts or wishes.

I am glad that you have settled my account with that troublesome gentleman, Mr. Denew, and I wish you would mention to Mr. Paytherus the Landlord's property Tax to see that it is right.

And now, if you please, let another account be settled. The fault lies at your own door. The lease you speak of was among the papers you have heard me mention as making their exit from my house at Berkely duriug one of my long visits to town. I dont quite give up its recovery and the other papers of great value such as Bonds, etc. I rejoice at your intelligence respecting America. How is General Lyman? I hope he weathered the severe winter well and that he does not suffer from our uortheastern blasts.

I have written to Mr. Addington I reply to a letter of his respecting the R. I. Society. My opinion I hope will meet their wishes. Shall I visit Mr Shipwith? His commendation would be useful. Mrs Jenner begs her compliments. Pray present mine to Mrs Murray and believe me

Truly yours, EDWARD JENNER.

P. S. As it is possible that there may still be a few remaining copies of your late publication what think you of your presenting some to the real friends of the vaccine cause, Lord H. Petty, Mr Windham, Mr W. Smith, etc, etc. The appendix should be marked so as to attract atteution as they may not perhaps like to go into the controversy. This alone would make any man laugh at the new vaccine trial.

BERKELEY, April 30th 1809.

[Jenuer Letter, No. 7, to Chas. Murray.]

Nov. 21, 1809.

MY DEAR SIR: Your last letter was truly doleful. On the one side the tragical details of the barbarous murders committed by the smallpox, and on the other hand, what to you is still more tragical, the unhappy tidings respecting your poor nephew. Be assured I sympathise with you most sincerely. I had promised myself the pleasure of seeing this young man, and hearing the history of his extraordinary life. But how seldom are our hopes and expectations realized. Just as we think them within our grasp, they vanish like a shadow.

The new institution cannot possibly succeed according to its present plan There is no energy in any one department of it. You must recollect how often I harangued the Board and Council of the B. I. Society on the necessity of making vaccination and all its benefits known to the people through the medium of the newspapers. And this was adopted by them as far as their means would allow. In my conference with Mr Rose I enforced, with all the powers I was master of, his making an adequate provision for this purpose and that it should be the duty of a secretary to attend to this point under the control of a superintendent meaning to take that office upon himself. But this you find has not been in the least attended to. Such a map of evidence, both foreign and domestic, might thus have been placed before the people that the destructive engine, smallpox inoculation, now kept so constantly at work would have no longer known its effects, and the stations of the N. V. Institution would have been crowded with applicants. But now all the documents laid before them consist in little more than the placards of Moseley and Birch, pamphlets, reviews, etc, etc, which abound with praises of the vaccine fall into the hands of a few only; but newspapers are read or their contents made known to the multitude. By the way, can you inform me who are the conductors of that excellent work the Annual Medical Register, one volume of which has been yet published? or what is more important to me, can you tell me the author of that part of it which relates to vaccination? It occupies thirteen closely printed pages and is executed in a style as masterly as what appeared in the Edinburgh Review on the same subject. Whoever may be the author he is fully entitled to my warmest acknowledgements. I never received Mr Brigg's publication. Anything of this kind will reach me if consigned to the care of Harvard Bookseller in Russell Street. Dear Sir Very faithfully yours,

E. JENNER.

[Jenner Letter, No. 12, to Charles Murray.]

Nov. 19, 1812.

MY DEAR SIR: I have not heard anything further respecting the horrid slaughter in the metropolis from the smallpox since your last tragical report. Your next, I trust, will be more favorable, but I shall dread to look at it. I have not heard from Mr. Winsor since you wrote and have unfortunately mislaid your letter, but if I recollect clearly Winsor was to let me know the new Resolutions of the Committee with respect to the limitations they intend to name for the old subscribers to enlarge their subscriptions. Should not a matter like this be advertized in all the public newspapers, or, at least, the most popular? My paper is the Globe, and a very popular one it is, yet no advertisement has ever appeared there.

Your friend Mr. Rigby and I have had some correspondence in consequence of what was communicated to me through you. I find he was a little out of humor with me for supposing some years back that he was

unfriendly to vaccination, I took up the idea from information made to me in a positive manner, but certainly did not remain long under this delusion as both his sister (Mrs. Parry) and yourself a long time ago convinced me of his attachment to the practice. Therefore I ought to be exculpated from lying under the influence of a temporary deception. I have told him that I know not which to admire most his philanthrophy or the ingenuity with which he wore it. The means which he employed for stopping the progress of the smallpox in Nowwich was a master stroke of good policy. What say the Board to this? I hope they have seen Mr. Rigby's Appendix. This paper should be put into general circulation.

I was shocked at a letter I received abont ten days since from the Rev. Mr. Pattison, the clergyman of Chertsey, informing me that the poor of the parish, consisting of some thousands, were immediately to be inoculated for the smallpox. He had the good fortune to modify the decree of the vestry and allow the poor creatures an option of smallpox or cowpox? Under the power of that spell which the abominable wickedness of Birch is still directing through the public prints, I fear very few will escape and embrace the latter. I have written very fully to their humane pastor, Mr. Pattison. The appendix sent to this gentleman, if it reached him quickly, would be well placed. In the midst of the storm I hope that you have put an umbrella over the head of Mr. Grant. With best wishes to you and Mrs. Murray, believe me very truly yours, E. JENNER.

[Jenner Letter, No. 13, to Charles Murray.]

July 25, 1813.

MY DEAR SIR: I wish you would have the kindness to tell me what sensation has been occasioned in town by the very extraordinary and unwarrantable remarks of my Lord Ellenborough in the House of Lords on vaccination? In the country they have occasioned no inconsiderable effect, and I will venture to predict without the interposition of some bold and manly counteraction the consequences will be extremely mischievous. The people who conclude without reasoning will imagine that a Lord Chief Justice knows everything; and judge that a general tremor must arise among those whose children have been vaccinated to find that they have received temporary security only and to hear the general slur his Lordship passed upon the practice that it did not merit the praises passed upon it. That it was very well for those who like himself had large families to bring up in a great city, etc. I have examined three different London papers and find the same expression in all of them; therefore I conceive his words to be correctly reported; but I imagine it will be necessary that a Report sanctioned by the House should be examined before what I am going to mention is published. My friend Dr. Baron, the fonnder of the Gloucestershire Vaccine Association, has written a most excellent letter full of the most spirited remarks and severe animadversions on Lord Ellen-

borough's conduct. It is addressed to his Lordship. I wish that you could confer with Mr. J. Moore on this subject and provide both printer and publisher. Dr. Baron is known to Mr. Moore, and you have seen his address to the Medical Practitioners of this country on the subject of vaccination, which alone is sufficient to evince his literary talent. It must also be duly advertized and all expense will be defrayed by myself. The title is "A letter to the Right Honorable Lord Ellenborough in reply to the observations on vaccination as reported in the public newspapers to have been delivered by His Lordship in the House of Peers on of 1813. By J. Baron, M.D."

[These spaces are left blank in the letter.—S. W. M.]

Pray have the kindness to let me have the result of your conference as soon as possible and a correct statement of what Lord E. said in the House.

Yours very truly, EDWARD JENNER.

[Postscript to Letter No. 13.]

The letter of course will be submitted to the eye of Mr. Moore and yourself before it is published, and if you think it necessary it may be laid before Council. I should be glad to have as many copies of the report of the N. V. E. as can be spared.

[Jenner Letter, No. 14, to Charles Murray.]

Aug. 29, 1813.

MY DEAR SIR: By this day's post I received my newspaper at Cheltenham, which is a certificate of your having received my letter addressed to you through the N. V. E. My good friend Woods might safely have hazarded his little monosyllable in reply to the assertion of the Collector of the London Vaccine Institution. Within these few days I have received a letter from Dr. Bradley saying that the R. I. S. is to be resuscitated and incorporated with the London V. Inst., and expressing an ardent wish (as the organ of the late meeting, the Doctor having been called to the Chair) that I would give encouragement to this new organization and become one of its members in being placed at the head of the Presidents. I have just been writing to your director and given the doctor's letter more in detail expressing at the same time a wish that he would obtain an early conference with you on the subject. As soon as this has taken place I hope to have your joint opinions upon this very extraordinary business. Not to accept this high honor was a point I determined on the instant it was offered. My time would have been thrown away even in hesitating on the matter. But don't talk of it till I have sent them my answer which they seem in a hurry to possess, and, therefore, I must beg you to be as early as your time will permit in conferring with Mr. Moore and letting me know the result of the conference. You may mention it privately to Mr. Woods. Is John Walker still one of the Society?

I shall send an order to my bankers to pay my arrears of subscription to the different institutions I support.

Do you recollect my asking you a long time ago to discover if you could the cause of my old friend Mr. Ring's coolness to me? It amounts to more than coolness indeed now—the cut complete. I have not the most distant conception of the cause, being unconscious of ever having given him the least offense, but on the contrary always showing him all the friendship in my power. It would be a great satisfaction to me to know the cause of this change of conduct. I feel assured it is imaginary. My best wishes attend you and your family.

Very truly yours, EDWARD JENNER.

Did you ever send a report to Mr. Rigby?

[Jenner Letter, No. 15, to Charles Murray.]

March 11, 1814.

MY DEAR SIR: I presume you have not been able to get the pamphlet looked over by Sir Edmund Carrington or any other of my learned friends and I think it hardly worth while now to take any further trouble about it. It is a sharp thing aud would have stung the Right Honorable gentleman pretty deeply, but I fear there would have been a reaction that might have been destructive to the excellent man who in the warmth of his heart produced it.

Many thanks to you for the oysters. So great a rarety as an oyster barrel seldom reaches me now. It is become about a triennial sight. I must come among you and let my friends know that I am alive.

How goes the National Vaccine Institution? You have been well cudgeld by the Edinburg men in oue of their late publications and I have very little pity for you. By this time I imagine you have a new Board. Can anything equal this in absurdity? Just as they are initiated in the business they are discharged and a new set fill their places who know nothing of the matter. Being self implicated in the transactions in the eyes of the public I must endeavor to clear my character as well as I can.

The last Report of the French Vaccine Committee is very excellent. They have sent it to me but if it once gets into Leicester Square it will never get out again Experieutia docet.

How singular it is that among all my London friends whom I have been endeavoring for these two years to prevail upon to execute a little commission for me in New Street Hanover Square no one should have hitherto accomplished it. Honest John still remains mute, but why, as I have told you before, I am unable to guess being conscious that I have never done anything to merit his frowns. Can you tell me what was the next move that Joseph Leaper made after his failure in endeavoring to draw me into the trap of the London Institution? I have heard nothing of John Birch lately.

I hope Mrs. Murray and your family are well. We want the return of Spring in the country to put an end to our colds. I have made enquiry respecting the late fatality of the measles through districts which when united form a population equal to that of Glasgow and the result is excessively against Dr. Watts hypothesis.

Very truly yours
EDWARD JENNER.

[Jenner Letter, No. 17, to Charles Murray.]

23rd June 1817.

MY DEAR SIR: Enclosed is a draft on Messrs Sadbroke for one hundred pounds.

I must confess I had no notion that I had incurred a debt of such great magnitude as your statement points out. You speak of interest and some further additions. Pray have the goodness to send me the whole amount. This once opulent country is in a state of ruin. The wretched condition of the agricultural laborer and the inability of the farmer to pay his rent, who can scarcely pay his taxes, have produced changes that are deplorable and were the manufactories to fall off below their present standard I fear the spirit of the people would occasion a general tumult. There is, thank Heaven, a prospect of general plenty in the fields and if we can hold up until the earth gives its iucrease things may mend.

The report you made to me from a French journal, which I have since seen, was too contemptible to require any reply from me. These jealous people still continue their ridiculous claims and now say the disease was known iu France twenty-five years ago. What nonsense? But the most extraordinary part of the business is that a name so illustrious as that of Chaptal should appear among these fellows. This I cannot understand unless what I have heard remarked is an absolute fact that all Frenchmen are alike.

Yours very truly,
EDWARD JENNER.

[Copy of letter addressed to his Excellency, The American Minister, Blakes Hotel, Jermyn St., London, by Edward Jenner.]

CHELTENHAM, Jan. 23, 1806.

DEAR SIR: I have been favored with your Letter from Bath and was sorry to find Mrs. Monroe had experienc'd a return of her indisposition. I know not why it should be so, but yet I see clearly that these febrile Catarrhs, which have obtained the name of Influenzas, hang about the Constitution for a long space of time, and that their influence, tho apparently subsided, is ready to be again and again, called into action from slight causes.

I mention this on Mrs. Monroe's account that she may guard herself with

more than ordinary care, till the exciting cause whatever it may be has quitted our atmosphere.

The Packet of Books for the worthy President I have desired a Friend in Town to consign to your care at Blakes Hotel, with all possible despatch. My Letter will be put up with the Book which is set apart for his own use.

And now my good Sir, allow me to thank you for all your kind attentions; and to assure you how extremely happy I am in having form'd your acquaintance and obtained your friendship. You will at all times find me ready to convince you of it in any way in my power, either on this, or the other side of the Atlantic.

With best wishes to Mrs. Monroe and your Family, I remain, Dear Sir

Your obliged and very faithful
humble serv't
EDWD JENNER.

THE REGISTRATION OF TUBERCULOSIS.

By LAWRENCE F. FLICK, M.D.

[Read April 4, 1900.]

The first step in an orderly scheme for the prevention of tuberculosis is registration. This is so self-evident that it looks like a waste of words to say anything in its favor. Yet, strange to say, there has been a storm of opposition within the ranks of the medical profession itself to its inauguration. Why has this opposition existed?

A careful review of the various and many arguments which have been made against registration seems to warrant the conclusion that the opposition has been largely due to fears and prejudices born of erroneous ideas about the contagiousness of tuberculosis. Some of these ideas are the outgrowth of traditional views, and others are the illegitimate offspring of the new science of bacteriology. The theory of heredity of tuberculosis and the old-time views about the etiological relationship between cold, want, grief, exposure, etc., and consumption still befog the mind of the average physician whose medical education antedates the last decade, even though he has accepted some of the new views which bacteriology has forced upon him. He admits that consumption may be contagious under certain circumstances, and that contagion, indeed, may play some part in the etiology of every case, but he sticks to his old fetish and believes that heredity plays the greater part, and that if it were not for cold, want, grief, exposure, etc., contagion would after all amount to nothing. He admits the existence of the tubercle bacillus because he can

see it, or because others in whom he has confidence can see it, but he looks upon it as an effect rather than a cause, and if a cause at all, merely a secondary one. Of the physicians who no longer worship at the old shrines a fair number are over-zealous converts to the teachings of bacteriology who have allowed their imaginations to run wild. They calculate the number of consumptives in the land, the number of bacilli which each consumptive expectorates per minute, per hour, and per day, the aggregate number of bacilli which must exist in the land; they expatiate upon the presence of the tubercle bacillus everywhere, in the house, on the street, in church, in school, in street cars, in railway cars, in sleeping-berths, indeed in every spot inhabited by human beings; and the medical skeptic, who also has a place in our professional ranks and who has not given much thought to the subject of bacteriology, with good horse-sense, concludes that, if all this is true, consumption cannot be contagious, for if it were everybody would have to die of it.

Thus while we have a passive adhesion to the doctrine of the contagiousness of tuberculosis by the medical profession, the attitude of the profession is more that of assent than of understanding; and not until the entire medical profession has a clear-cut idea of what the contagiousness of tuberculosis really means can we hope to have unanimous support of the profession for registration; and until we have unanimous support of the profession we cannot hope to overcome the natural repugnance of the people at large to what they are disposed to call unnecessary espionage upon the privacy of domestic life. For the discussion of registration we must therefore enter upon the subject of contagiousness of tuberculosis.

Tuberculosis is a contagious disease and not an infectious one; and right here we have the first bone of contention and source of misunderstanding within the profession, even among the well-informed. Owing to the loose manner in which the words contagious and infectious have been used in medical literature, a great deal of confusion has arisen. A contagious disease is one in which the disease-germ is given from one

host to another without passing through an intermediary host or culture-medium; or, in other words, a disease which is due to an organism that usually does not reproduce itself outside of the host upon which it maintains its parasitic existence. The parasite producing such a disease is always conveyed from one host to another either by direct contact or indirectly through fomites. An infectious disease, on the other hand, is one in which the parasite may have an intermediary host, or at least reproduce itself in an intermediary culture-medium. As an exemplification of the difference between a contagious and an infectious disease we may take smallpox and malaria. The germ which produces smallpox is always conveyed either directly from one host to another, or indirectly through some fomites, such as clothing, articles which have been handled by a host, or a room which has been occupied by him. So far as we are able to judge from clinical data the germ which produces smallpox does not reproduce itself outside of a host, and, although it may maintain its existence for some time during the interval of passing from one host to another, when it does pass it enters the new host in the same generation in which it took its exit from the old. For the prevention of smallpox, therefore, all that is necessary is to prevent the host from coming in contact with susceptible persons and to properly sterilize all fomites with which he has been in contact. On the other hand, the organism which produces malaria does not pass directly from one host to another, nor indirectly through fomites. It does not even necessarily pass in the same generation, but after reproduction in an intermediary medium, such as water, soil, or a host of another species, may pass into the new host in a generation far removed from the organism which was given off from the old host. In the prevention of malaria, therefore, we have to go beyond the patient and his environments to the intermediary medium or host if we wish to accomplish our purpose. Other examples of contagious diseases are scarlet fever, measles and chicken-pox, while typhoid fever, yellow fever and cholera are other examples of infectious diseases. Malaria is an in-

stance of a purely infectious disease, while typhoid fever is an instance of a disease which may probably be contagious as well as infectious. There is more than an academic difference between a contagious and an infectious disease, since in a contagious disease pollution is limited and can only reach persons who come in contact with the source of pollution or with something which has been in contact with it, whereas, in an infectious disease, pollution must be general, and may reach any member of a community without contact with the source or with anything that has been in contact.

Tested by the diseases which are types of contagious and infectious diseases, tuberculosis is strictly a contagious disease; for the bacillus has usually no intermediary cycle of life or stage of existence, but goes directly from one host to another. When it is given off from a tuberculous subject it may maintain its existence for a long time in the necrosed tissue in which it is thrown off, but its cycle of life is suspended for the time being, and can only be resumed when it finds lodgement in congenial soil, which is usually a new host. It of course may find congenial soil, and all the conditions necessary for propagation of its species in an artificial culture-medium, but in practical life such a medium is not likely to be supplied to it except when specially and intentionally prepared for it in the laboratory. Even in the laboratory there is a limit to the number of artificial mediums in which it will grow. In its transmission from person to person the necrosed tissue in which it is given off plays a very important rôle. This tissue dries up and becomes pulverized, and in a fine, dry state cachés the bacillus, so to speak, until chance again carries it into a new host. So long as the bacillus is protected by the necrosed tissue it maintains its viability and is capable of taking up its cycle of life in a new host, but when the elements reduce the necrosed tissue to elementary principles and release it from confinement it speedily succumbs to the germicidal powers of light and air. In practical life the tuberculous subject is, therefore, most dangerous to those in his immediate environment, and is apt to contaminate the room which he

occupies, the clothing which he wears, and the utensils and implements which he uses. He also may contaminate articles of food which he handles. The greatest danger to the well is, therefore, along a line of social relations, namely: first, from intimate contact with the sick; secondly, from occupancy of contaminated rooms; third, from use of contaminated utensils, implements and clothing; and fourth, from use of contaminated food. By whatever method the disease may be conveyed necrosed tissue plays an important part, and contact, direct or indirect, is necessary. In every sense, therefore, tuberculosis is contagious, and in no sense is it infectious.

There is, of course, an individuality and a limitation in the contagion of tuberculosis. It will not do to base our ideas of the contagiousness of tuberculosis upon an abstract idea of contagion, or upon our conception of the contagiousness of smallpox, measles, scarlet fever, or of any other of the contagious diseases which long since have been recognized as contagious and for which measures of prevention have been established. Smallpox, measles, and scarlet fever, as, in fact, all the exanthems, are intensely contagious, because their contagion is given out from all parts of the body, and contaminates everything in the immediate vicinity of persons suffering from them. They are, moreover, very acute diseases, having short incubation-periods, and running their courses in comparatively brief periods of time. Their contagious nature is so manifest as to inspire fear and apprehension, leading to extreme and radical measures for their prevention. They have practically given form to the abstract idea of contagion, which makes it very difficult to elucidate the concrete idea of the contagiousness of tuberculosis or of any other contagious disease which differs radically from them. Even in the professional mind the contagiousness of a disease is measured perhaps unconsciously by them. The contagion of tuberculosis is given off through a single channel, and that a restricted one, namely, the necrosed tissue, in the form of sputa when the disease is in the lungs, and in the form of pus when the disease is in some other part of the body. Tuberculosis runs a very slow course,

as a rule, having a long incubation period and passing through many series of attacks. Inasmuch as the tubercle bacillus cannot maintain its viability long without the protection of necrosed tissue, and inasmuch as necrosed tissue, by reason of the repugnance which it inspires is likely to be disposed of in some way or other immediately upon being thrown off, the chances of contamination of the environments of the patient are very much less than with the exanthems for an equal length of time. Were the duration of tuberculosis no longer than that of smallpox, measles, or scarlet fever, it is probable that the disease would long since have run itself out, as the contamination of the environments of the patient would have been too trifling to have kept the disease alive. But what is lacking in intensity in tuberculosis is made up, for in duration and the contamination of the environments by gradual increment probably become as intense as with the exanthems by the time the end of the disease is reached. The chief difference, therefore, from a practical point of view, between the contagiousness of tuberculosis and that of the exanthems lies in the time required for contamination of environments and fomites to a potency capable of transmitting the disease to others. Momentary contact with smallpox may convey the disease, but it requires prolonged contact to convey tuberculosis. With the exanthems a room which has been occupied but an hour, an implement or utensil which has been handled but once, and food which has been merely in the proximity of the person affected may convey the disease to others, because in that brief period of time contamination may have become intense enough to give a successful implantation in new soil; while with tuberculosis it is necessary that a room shall have been occupied for a considerable time, that implements and utensils shall have been either used a great deal or smeared with necrosed tissue, and that food shall have necrosed tissue mixed with it in perceptible quantity before successful implantation in a new subject is likely to take place.

The relative potency of contagions, or the degree of contagiousness of diseases, can probably best be measured for

practical purposes by the number of successful implantations which take place in a given period of time, provided that the time be made long enough to allow for idiosyncrasies of the diseases compared. In a comparison of tuberculosis with the exanthems it is necessary to take a long period, because of the very slow progress of tuberculosis, the long time required for the establishment of efficient contamination, and the mild immunity which the disease is capable of setting up on the one hand; and the rapid course of the exanthems, the speediness with which they produce efficient contamination, and the strength and durability of the immunity which they set up on the other. Twenty years, or the average period of a generation, probably would be a fair length of time for such a comparison. If we thus compare the contagiousness of tuberculosis with that of scarlet fever, which is admittedly one of the most contagious diseases, we will find them of nearly equal potency. For example, there were in the city of Philadelphia during the period from 1868 to 1888, 51,014 deaths from consumption, which is one form only of tuberculosis, and 9024 deaths from scarlet fever. Admitting that 75 per cent. of all cases of tuberculosis die, which is, I think, an overestimate, and that only 10 per cent. of all cases of scarlet fever die, which is probably an underestimate, we will have had in Philadelphia during that period 68,018 cases of tuberculosis and 90,210 cases of scarlet fever. If we would add other forms of tuberculosis to the cases of tuberculosis of the lungs, we would probably find that the number of successful implantations of tuberculosis equalled and possibly exceeded that of scarlet fever. What is true as between tuberculosis and scarlet fever is equally true as between tuberculosis and the other exanthems.

Objection will no doubt be made to this comparison by men who admit the contagiousness of tuberculosis, but claim that contagion is only one of many factors which enter into the etiology of the disease. But is such an objection logical? Whatever factors may enter into the etiology of tuberculosis besides contagion there can be no doubt about contagion being

the only essential factor. It is no more possible for tuberculosis to develop without contagion than it is for scarlet fever to develop without contagion. Both are due to a living organism, and this being so the chain of contact between cases must remain unbroken, unless it can be shown that the organisms have an intermediary life in a culture-medium or in a host of another species. A disease cannot be contagious and non-contagious at the same time. If it is due to organic life it must be contagious or infectious, and can be nothing else; and if it is not due to organic life it can neither be contagious nor infectious under any circumstances. If we admit at all that tuberculosis is due to organic life and is contagious, logic compels us to admit that every case of tuberculosis which arises has sprung from another case. Exposure, want, grief, overwork, close confinement, malnutrition, etc., may act as predisposing causes, but they cannot produce the disease without the tubercle bacillus and without contagion. Neither can we say that the factors just enumerated do not play an equally important part in the etiology of scarlet fever. It is probable that a low state of vitality, by whatever cause produced, predisposes to disease caused by organic life; but, if it is true, it is true as a general proposition, and applies to all diseases of this character alike. Lowered vitality is, however, not the only predisposing cause of parasitic disease. Even a stronger predisposition may be found in family and racial proclivities, and this applies as well to tuberculosis as it does to the exanthems. But it would lead too far afield to go more deeply into the questions of predisposition and acclimatization in this connection. Whatever modification they can legitimately bring to our ideas of contagion must apply with equal force to all contagious diseases, and can have but little bearing upon the question of the registration of such diseases for the purpose of prevention.

Registration of the exanthems has long since been practised. The justification of it lies in the right of the government to protect the subject against avoidable danger to life; the reason for it lies in the necessity for government control of (1) con-

tact between the well and sick and (2) proper sterilization of fomites. Now, what is there in the justification and rationale of the registration of the exanthems that does not apply with equal and even greater force to tuberculosis? The exanthems set up an avoidable menace to life, so does tuberculosis, but with this difference: tuberculosis is more amenable to preventive measures than any of the exanthems except smallpox, and can much more readily be stamped out of civilized communities. With the exanthems control of contact between the well and sick is necessary for the purpose of prevention; the same is true of tuberculosis, only that with tuberculosis the control must be of a different character. With the exanthems control, as exercised at present, means quarantine of the subject of disease. This is accomplished in two ways: (*a*) by removing the patient to a hospital under quarantine and (*b*) by putting a placard on his house to warn the public against danger, and at times even putting a guard around it to prevent ingress and egress. Whatever may be said in support of these harsh, odious methods of controlling contact between the well and sick in the exanthems, they are certainly unnecessary for the control of contact between the well and sick in tuberculosis. Control of contact does not necessarily mean estoppel of contact, but may mean regulation of contact; and regulation of contact may be a much more efficient way of stopping the spread of disease than estoppel, because it is more humane, and is, therefore, more likely to have the co-operation of the afflicted. For the control of contact in tuberculosis removal of patients to hospitals without quarantine would in most instances be the most practical and at the same time the most humane method. The slowness of the disease and the hopefulness of the patient lend themselves well to such a method. Tuberculosis usually runs a considerable time before the patient begins to give off contagion. During this time the keen edge of sorrow at being stricken with disease is worn down somewhat, both for the patient and the family, and the matter of having the patient go to a hospital can be viewed more calmly than it could have been in the beginning. The

hope of recovery, which lingers to the last in the breast of the consumptive, likewise makes for willingness to go to the hospital, and this will be true to a still greater extent when it once becomes generally known that hospital treatment gives the best chances of recovery. If sanatoriums were established in the mountains for incipient cases, and hospitals in or near large centres of population for advanced cases, many would seek admission, and the majority could readily be induced to enter. In the sanatoriums and hospitals patients could very soon be taught to make themselves innocuous by the observance of certain rules for the disposal of necrosed tissue. Briefly stated, such rules would be, first, that all necrosed tissue be sterilized immediately upon being given off, by ejection into a liquid containing a germicide; and, second, that paper napkins be used to wipe the lips, and that they be burned. With the observance of these rules and with scrupulous cleanliness of hands and finger-nails, patients could be permitted to have free intercourse with relatives and friends, thus relieving the measure of the harshness of quarantine.

For tuberculous subjects declining to enter sanatoriums or hospitals explicit rules could be laid down, dealing in all detail with the sterilization of necrosed tissue and the maintenance of personal cleanliness. Among the intelligent and well-to-do the enforcement of such rules could safely be entrusted to the family physician, while with the ignorant and poor it would have to be assumed by the Board of Health. A proper penalty for non-observance of the rules would be commitment to a sanatorium or hospital. Legal authority, supplemented by kind, sympathetic and persistent effort at instruction, together with such material aid as would be necessary among the very poor, could so regulate contact between the well and sick, in even the humblest home, as to practically rob it of danger.

The second part of the reason for the registration of the exanthems is the proper control of the sterilization of fomites. This part has even stronger foundation in tuberculosis than in the exanthems. The contagion of the exanthems, as well

as that of tuberculosis, is kept alive during the interval between hosts by necrosed or decayed tissue, but in tuberculosis the necrosed tissue is much grosser in character than in the exanthems, and, therefore, its devitalization is more difficult. The methods which have been found efficient in the sterilization of rooms which have been occupied by exanthematous patients are inefficient for the sterilization of rooms occupied by consumptives. Nothing short of the most searching cleansing, by which every particle of necrosed tissue is removed from the room, will suffice for the sterilization of a room which has been occupied by a consumptive. Such a cleansing requires the supervision of an expert, and is likely to be too expensive to be given voluntarily by either the tenant or the landlord. Anything short of it is a mere placebo and a snare to lure the unwary to their doom.

In practical life, as matters stand at the present day, fomites are a very frequent means of conveying tuberculosis from the sick to the well. Next to intimate prolonged contact between relatives they constitute the most frequent means. Family relationship gives rise to probably 50 per cent. of all successful implantations of tuberculosis; house contamination to probably 25 per cent; and contamination of clothing, utensils, implements, and food to from 15 to 20 per cent. The remaining cases, ranging from 5 to 10 per cent., are probably implanted through chance contact with tuberculous subjects, through the inhalation of contaminated dust in public places, in street cars, sleeping-berths, and through the use of tuberculous milk and meat, etc. If anyone has doubts about the approximate correctness of these figures, let him but take the trouble either to trace carefully a given number of cases of tuberculosis to their origin, or to inquire into the histories of all the cases of tuberculosis which have occurred in a given district in a given time, and he will certainly convince himself of their correctness. In 1889 I made a careful study of all cases of tuberculosis which ended in death during that year in the Fifth Ward of the city of Philadelphia. Of 83 cases of probable tuberculosis about which I was able to get definite

information I found that 48 had died in houses which had been occupied by tuberculous subjects before, and that of the 48 only 5 bore the same name as the former occupant. Of the 48 cases 12 had the disease when they moved into the houses in which they died, but 4 of these I was able to trace to former contaminated houses. Of the 35 patients that died in uncontaminated houses I found that 7 had the disease when they moved into the houses in which they died (of these two had lived in contaminated houses before they moved into the houses in which they died); 5 had lived in lodging-houses, 2 were associated with consumptives in business; 1, although reported from an uncontaminated house, had really died in one, and 1 had died of syphilis. Of the 83 cases there were only 19 in which I was unable to trace some sort of exposure to contagion, and in the 19 I would probably have been able to discover exposure if I could have consulted the victim himself. One of the most surprising facts which came out in this investigation was that only 5 persons out of the 48 who had died in contaminated houses bore the same name as the preceding victim in the same house. Standing by itself, this fact would indicate that house-contamination should change places with family relationship in the table of etiological factors in tubercular implantation; but clinical observation will not tolerate such a conclusion. It must, moreover, be borne in mind that the name is in itself an unsafe indication of what part family relationship plays in a case, inasmuch as among the poor, relatives of different names frequently live in the same house in intimate relationship.

In the beginning of my thesis I laid down the proposition that the first step in an orderly scheme for the prevention of tuberculosis is registration. The major of the premises of the syllogism of which that proposition is part of the conclusion is that tuberculosis is a contagious disease. I have endeavored to show that tuberculosis is a contagious disease in the true and technical meaning of that word, and not an infectious one. If I have done this I have finished my thesis. It may not be amiss, however, to say a word about the waste of energy

which is being expended for the prevention of tuberculosis on mistaken grounds—energy which could be so much better expended in the right direction—and about the groundless fears which are being excited in the public mind by overzealous but badly-informed contagionists. Much effort has been made and is being made daily throughout the country to have anti-spitting laws passed and enforced; agitation is going on to have laws passed prohibiting marriage between tuberculous subjects and to tuberculous subjects; hundreds of thousands of dollars are being spent for the detection of tuberculosis among dairy cattle and for the destruction of tuberculous dairy cattle as a measure for the prevention of the spread of tuberculosis among human beings. All of this effort is misdirected so far as the prevention of tuberculosis among human beings is concerned. Spitting is a vile habit, but to the consumptive it is a necessity, and with him the remedy is to teach him how to spit without injuring others. Spitting in public places, in street cars, and upon sidewalks should be prohibited for the sake of cleanliness and decency, but not on the score of preventing the spread of tuberculosis. The tuberculous sputum which is ejected out-of-doors, whether it be on the sidewalk or in the streets, and even that which is ejected into street cars and in public places such as public buildings, will probably not account for five per cent. of the implantations of tuberculosis. Sunlight, air, and water are mortal enemies of the tubercle bacillus, and in the majority of human beings prolonged exposure is necessary for a successful implantation of tuberculosis In this connection Cornet's investigation into the death-rate of the street cleaners of Berlin should be borne in mind. The street cleaners of Berlin hold their positions for life, and although they daily inhale the dust from the streets they show one of the lowest death-rates from tuberculosis of any class of workmen in Berlin. So far as I am able to judge from a somewhat extensive experience with tuberculosis among the working classes and the poor, the street car conductor is also comparatively exempt from the disease. Consumptives should never marry, not, however, because the

spread of tuberculosis would thereby be prevented, but because married life is, as a rule, non-conducive to recovery from tuberculosis. When a tuberculous subject has recovered there is no longer an impediment to his marriage, for not only is tuberculosis not hereditary, but the probabilities are that tuberculosis in the parent tends to set up an immunity in the offspring. The extermination of tuberculosis in dairy cattle is highly commendable as an economic measure, but not as a measure for the prevention of tuberculosis in human beings. My conviction is that while tuberculosis is frequently conveyed from human beings to animals, it is rarely conveyed from animals to human beings. Spitting into grass and into food of domestic animals by consumptives is probably a prolific source of tuberculosis in the brute creation, and herein anti-spitting crusaders should seek justification of their labors. The scares about the contagiousness of tuberculosis which are set loose upon the public by badly-informed extremists are as cruel as they are unfounded. Mere casual contact with a consumptive is not dangerous, and even intimate, prolonged contact can be made innocuous. If these truths were but generally known and understood, how much easier would be the life of the poor consumptive! There is no necessity for turning our backs upon the consumptive poor—no justification for turning them out-of-doors. We can teach them how to make themselves harmless, and we should do it: but the WE upon whom this duty devolves is our representative, the government. What is everybody's duty is nobody's duty unless the government takes it up, because a people cannot perform a universal duty except through its properly constituted head. But in order that the government may be able to do its duty to the consumptive it must first know who and where he is, and this knowledge can only be obtained through registration.

Registration of tuberculosis is necessary in order that (1) family relationship can be controlled by the government so as to make contact between the sick and well harmless; (2) that contamination of fomites may be prevented, and when it occurs that the contaminated fomites may be properly steril-

ized; and (3) that the contamination of implements and food may be prevented. These results can only be made attainable through registration, and by their attainment 90 per cent. of all successful implantations of tuberculosis may be combated. How short-sighted to soothe ourselves into a sense of security by a fussy explosion of energy in measures which at best can only control perhaps 10 per cent. of implantations, while we disregard much simpler and more humane measures which promise fair to exterminate the disease in a short time.

If anything further need be said in favor of registration it is that through registration alone can the thousands and tens of thousands of families who annually change their places of abode, and the hundreds of thousands of individuals who frequently change their lodgings, be protected against the danger of contracting the disease in contaminated houses and rooms. What can be more unjust than permitting an innocent family or individual to move into a death-trap without giving them the slightest chance of discovering the danger and forestalling it? If every case of tuberculosis were registered the family or individual seeking a new place of abode would at least have it in their power by foresight and care to avoid a danger which the government should but does not remove from them.

DISCUSSION.

Dr. S. Weir Mitchell: Our friend says that this disease is not hereditary. I should like to ask exactly what he means. A great many of us know of families in which all of the members die of consumption.

Dr. Flick: The explanation of the phenomena which have been stated is, I think, to be found in the distinction between heredity and predisposition. In speaking of heredity of tuberculosis we must bear in mind that there may be a family predisposition which may run through an entire family tree, but heredity has to be decided by the effect of tuberculosis in the parent upon the offspring. The mere fact that various members of a family have died of tuberculosis—aunts and uncles, brothers and sisters, nieces and nephews—does not argue in favor of heredity. It is simply an evidence of family predisposition which exists in all. If the child of a man who was consumptive when the child was procreated shows afterward a

stronger predisposition to consumption than the father did, we may conclude that tuberculosis was transmitted from the father to the offspring; but if a weaker, that partial immunity was transmitted. I have now under observation a number of children who were procreated while one or other parent was tubercular, and I am thoroughly convinced that to a certain extent tuberculosis in the parent does produce partial immunity in the offspring, and that a child which has been procreated by a consumptive father or born of a consumptive mother is less liable to develop tuberculosis than the child of parents who have not had the disease. But even broader and stronger evidence in favor of the establishment of immunity can be found in the existence of racial predisposition and racial immunity to tuberculosis. The very fact that the races that are free from tuberculosis develop it in an extraordinary degree when they come in contact with it, and that races that have been in long contact show comparative resisting force, is to some extent a strong argument in favor of the existence of an immunity after prolonged exposure. Take, for example, as an illustration of these phenomena, the colored race, which, according to reliable authority, in the interior of Africa is free from tuberculosis, but when it comes in contact with the disease in this country or in Europe is exceedingly susceptible to it, so that 75 per cent. at least of all who are exposed fall victims. Also the American Indians, who, prior to their contact with the white man, were absolutely free from tuberculosis, and who, when they came in contact, fell a ready prey to the disease. The Caucasian race, on the other hand, which has struggled against the disease for centuries, shows a growing immunity. The reason why the Caucasian race is less susceptible than the colored race and the Indian race is because for centuries it has been exposed, and there is probably no family that has not had the disease at some time or other.

I believe that immunity thus established, while not permanent, is of the same character as in smallpox. In the course of centuries such immunity becomes established.

DR. MITCHELL: That the disease is directly inherited, of course, none of us hold, but that something exists in certain families which makes them fertile ground for consumption.

Dr. Hopkins requests me to ask why so often the disease is absent from one generation and appears in the next.

DR. A. V. MEIGS: I should like to ask Dr. Flick whether he has any explanation to offer of his statement that the child of a consumptive parent is less liable to develop consumption than the children of healthy parents, or if he wishes what he has said is to be understood by us as a statement of an opinion of his own based upon his clinical observations. If what he has said about the children of consumptive parents is true the fact is a most curious one.

DR. FLICK: In answer to Dr. Meigs' question I may say that I think there is an explanation outside of clinical observation as well. I have based

the statement largely upon clinical observation, but the idea of establishment of partial immunity in tuberculosis is, I think, in harmony with our knowledge of immunity established by all diseases due to organic life, and is in harmony with other phenomena in biology, such as the exhaustion of soil by organic life. This is illustrated throughout the entire vegetable kingdom.

I am not the only one who has made clinical observations upon this subject. I believe that when a more careful study has been made of the effect of tuberculosis in the parent upon the offspring we will get at the truth and be able to show that the general principle that all organic life has the power of establishing resisting power to parasitic life applies to tuberculosis as well as to smallpox and other diseases due to disease germs.

It is upon this broad principle as well as upon clinical observations that I make the statement.

In regard to tendency of certain families to tuberculosis, of course, Dr. Mitchell and I agree. Dr. Mitchell calls it family tendency, and I call it predisposition. I do not think anyone would dispute the fact that there is a family tendency to all diseases, but that is not the old idea of hereditary tuberculosis, which was that the disease was directly transmitted, and that because parents had tuberculosis the children were almost certainly doomed to die of it. I do not think such views are any longer consistent with our ideas of disease and of biology in general.

PRESENTATION OF A SPECIMEN OF OSTEOSARCOMA OF THE LEFT FEMUR REMOVED BY A HIP-JOINT AMPUTATION.

By ROBERT G. Le CONTE, M.D.

[Read April 4, 1900.]

This specimen was removed five days ago, and shows very well another form of sarcoma—the giant cell- or osteosarcoma. You see the marrow, the bone substance, and the surrounding muscular and fibrous tissue invaded by the new growth. The patient's history is as follows:

A. G., aged eighteen years, white, single, call-boy; family history negative. He has always been delicate, but the only disease he remembers having is measles. Last October (six months ago) he began to have pain in the left femur; was under treatment for rheumatism. January 3d he fell four feet, striking the left thigh. This injury greatly increased his pain, and was soon followed by a local swelling of the limb. The three characteristic points in this history are the age of the patient, the local pain in the thigh, for which no cause was assigned except rheumatism, and the swelling, being first noticed after some slight traumatism. During the year I have seen three other cases of osteosarcoma of the tibia and femur. The ages ranged from fifteen to twenty-one; each one had been treated for months for rheumatism, and two of the three cases attributed the swelling to some slight traumatism.

The swelling in the specimen before you begins about eight inches above the knee, and extends almost to the trochanter. The circumference is two and a half inches greater than the right thigh. A blood-count shows the red cells to be 4,680,000, the white 16,000, and the hæmoglobin 78 per cent.

The procedure of the amputation was as follows: An Esmarch bandage was applied from the toes to three inches above the knee and secured. While the limb was elevated the lower portion of the growth was incised

and a small portion removed for immediate microscopic examination. With the limb still elevated, Wyeth's pins were inserted and a piece of drainage-tube was made to tightly encircle the limb above the pins until the femoral artery had ceased to beat. A circular cuff of skin was dissected back to the great trochanter, where the muscles were divided and the head disarticulated. The moment the limb was removed, owing to the nearness of the elastic ligature, the muscles were drawn up and rolled inward, drawing the vessels with them. Had the section of the muscular tissue been a little higher, I feel confident that the pressure of the elastic ligature would have forced the femoral vessels above it, and disastrous hemorrhage would have resulted before the artery could have been secured. I would, therefore, advise, when the muscles have to be divided high up, that the femoral vessels be ligated or secured with a hæmostatic forceps before the head is disarticulated, or that some other procedure of amputation be used. The vessels were then secured, the muscles united with a few deep catgut sutures, to prevent in a measure the free oozing which always follows the elastic ligature, drainage inserted, and the skin closed with silkworm-gut. Time of operation, thirty minutes; loss of blood, slight; shock, slight.

PERIPHERAL RESECTION OF FIFTH NERVE.

THREE CASES WITH MICROSCOPIC EXAMINATION OF THE PORTIONS OF THE NERVES REMOVED AND REPORT ON THE LATER CONDITION OF THE PATIENTS.

By W. W. KEEN, M.D.,
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AND

WM. G. SPILLER, M.D.,
PROFESSOR OF DISEASES OF THE NERVOUS SYSTEM IN THE PHILADELPHIA POLYCLINIC, ASSOCIATE IN THE PEPPER CLINICAL LABORATORY, UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA.

[Read at a special meeting held April 20, 1900.]

Dr. Keen's Report.

Case I.—*Resection of supraorbital and infraorbital nerves for tic douloureux.* Mrs. A. K., aged fifty-three years, a patient of Dr. S. Weir Mitchell, first came to the clinic of the Infirmary for Nervous Diseases, February 11, 1898. She had always been healthy, and of her eleven children, six were living and well.

In October, 1894, she felt a sudden, slight tingling sensation at the internal angle of the right eye. This increased in severity and finally included the entire right face and anterior part of the scalp. She had to sit up, as lying down increased the pain so much. In 1895 she was very much better. In 1896 she had scarcely any pain at all, but on February 4, 1898, it suddenly recommenced, and she had suffered ever since. Along with the pain there was spasmodic twitchings of the muscles about the right eye, and of the face, as long as the pain lasted. On March 3, 1898, I removed the supraorbital and infraorbital nerves, the latter by the orbital route, dividing the nerve toward the apex of the orbit. On January 9, 1899, she again reported at Dr. Mitchell's clinic. She had suffered

no pain after the operation for eleven months, then had a recurrence for two months, accompanying la grippe. In the last two weeks she had had another recurrence, but the pain was neither severe nor constant.

Case II.—*Resection of the infraorbital and inferior dental nerves for tic douloureux after ineffectual treatment by massive doses of strychnine.* Mrs. M., Princeton, N. J., aged sixty-two years, first consulted me December 1, 1898, at the instance of Dr. James H. Wikoff. Twenty years ago she had a very serious hemorrhage from a uterine myoma, and a sister died after a hysterectomy for the same trouble. For ten years she had been a great sufferer from hemorrhoids, attended with profuse hemorrhage. She also has a femoral hernia. Ten or fifteen years ago, without assignable cause, a sudden partial palsy of the left side came on, but yielded to treatment. For some time she has had a slight rise of temperature toward evening, and has been more or less of an invalid. The uterine myoma still persists, but has given her no trouble of late. Three years ago, without assignable cause, she began to have excessive pain in the groove between the nose and right cheek. It has now extended over the whole of the cheek and the lower jaw and also to the ear. The pain is the characteristic pain of tic douloureux, and is caused by eating, conversation, etc.

Desiring, first, to test the effect of strychnine, which in several cases had enabled me to avoid operation, I commenced with $\frac{1}{20}$ gr. t. d., adding one pill to the daily dose, so that the first day she would take three pills; the second, four; the third, five, etc. When she reached six pills a day she was to add one pill every other day until she had reached $\frac{1}{2}$ gr. a day or until the pain had ceased. Up to the time when she was taking $\frac{7}{20}$ gr. a day no relief from the pain had been experienced, nor had any strychnine symptoms developed. Ordinarily I have found these symptoms develop slightly at first, so as to give warning, but in this case the onset was sudden. Stooping down to pick up something from the floor, her legs were suddenly violently extended and her body was thrown some ten feet across the room. She suffered no injury, as her husband fortunately caught her. The medicine was immediately discontinued, and she decided on operation.

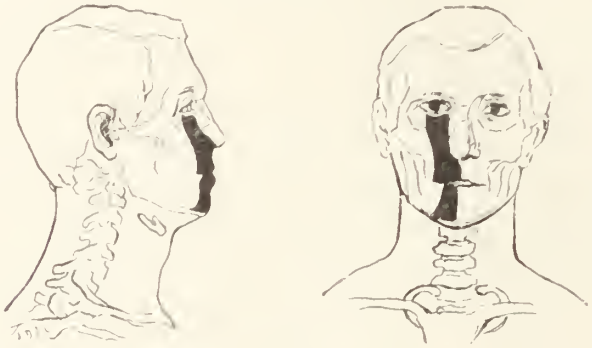
This was performed on December 31, 1898. By a slightly curved incision at the lower border of the orbit, the nerve was exposed in the orbit as well as on the face and 3 cm. of the infraorbital resected. I then trephined the lower jaw and removed 5 cm. of the inferior dental, including the mental and incisor branches. The canal in the bone at both ends was plugged with some of Horsley's antiseptic wax. She made a typical, smooth recovery, her temperature only once reaching 100° F., and she went home on January 13, 1899, two weeks after the operation.

Two days after her return she learned, by an operation on her husband, that he had an inoperable internal cancer. She nursed him through the operation and for some time afterward, when he died. She wrote me under date of February 19, 1900, that after the death of her husband she was obliged to build another home and move into it in September, 1899. This involved considerable planning, anxiety, and care. Up to that time she had been entirely free from the facial pain, but the moving caused great mental and physical strain, and a return of the pain followed for some weeks. Recently she has also had another attack of about the same duration, but when she wrote she was entirely free from it. In each instance, however, she had taken cold and suffered from much irritation of the throat and from cough. The pain seems to have been limited entirely to the lower jaw, and was especially provoked by conversation or eating. She lost no sleep, however, and by keeping very quiet the pain lessened and finally disappeared. It came in spasms with a sensation "as if her jaw were in a vise and being twisted." Her relief from the pain, by the operation, was so great that she writes she would unquestionably seek relief again by another operation should it prove necessary.

Concerning the examination of her face made by Dr. Spiller, January 10, 1899, after the operation, he says: "The anæsthetic area begins about a quarter of an inch below the margin of the right lower lid. Complete anæsthesia for touch, temperature and pain exists in the shaded area. (Fig. 1.) The anæsthetic zone on the side of the face is sharply defined, as it is also in the median line of the face below the nose. It extends over the right ala nasi. Tactile sensation in the right nasal passage is much impaired. Tactile

anæsthesia is present on the inside of the lips, on the outer and inner surfaces of the lower gum, extending from the median line of the face to about the angle of the mouth. The upper gum appears to be anæsthetic to touch on its outer and inner aspects from the midline of the face to about the angle of the mouth."

FIG. 1.



CASE III.—*Resection of infraorbital and inferior dental nerves for tic douloureux after failure of massive doses of strychnine.* Miss N., aged twenty-eight years, first consulted me in the early part of 1898, at the instance of Dr. Kinard, of Lincoln, Pa., for tic douloureux, limited to the infraorbital and inferior dental nerves of the right side. As in Case II., I wished to test the effect of strychnine before proceeding to operation. She began the treatment under Dr. Kinard's direction on May 9th, and continued it up to January 26, 1899. She began with $\frac{1}{40}$ gr. t. d., increasing one pill every day until she was taking $\frac{8}{40}$ gr. As she then began to feel some of the effects of the strychnine, the dose was not increased, but she took eight granules a day for five months. The strychnine was then increased to nine, and after a week to ten, a day. After taking $\frac{10}{40}$ gr. a day for two days she complained that she felt very nervous and weak, and the arms and legs trembled. The number was then reduced by Dr. Kinard, first to nine and then to eight granules up to January 26th. Finding that the strychnine was inefficient, she then desired operation.

She entered the Jefferson Medical College Hospital on January 27, 1899, when the following history was elicited: Her father and mother

were both living and well, between fifty and sixty years of age: also nine brothers and sisters. She had the ordinary diseases of childhood, including diphtheria, and made good recoveries, but no other illness till the present trouble began. Menstruation began at thirteen, and has always been regular but painful; occasionally she had leucorrhœa. There was no evidence of any venereal trouble. Nine years ago she began to have pain on the right side of the face. At first it occurred in brief paroxysms at long intervals, being apparently caused by eating food which was very hot or very cold, or by exposure to draughts of cold air. The pain began above the right ala of the nose, and was sharp and shooting in character. Gradually the paroxysms became prolonged and the pain spread over the entire right face, except the forehead, which has rarely been involved. For the last three years she has suffered almost constantly, though with slight lessening of severity for short intervals, but the pain has never disappeared entirely. The upper teeth on the right side were extracted three years ago and last spring the lower teeth, but without the least relief. When the pain is most severe she states that the sputum is tinged with blood and mixed with a slight amount of yellowish, thick discharge, and that there is a very marked overflow of tears from the right eye. The urine was normal.

On January 29th, Prof. H. A. Hare, at my request, examined her lungs and found an impairment of resonance on the right side. Respiration was so defective that nothing could be observed by auscultation. Examination of the right ear, by Dr. Klopp, gave a negative result. Dr. Jones also examined her mouth and throat and found only a slight catarrh. Dr. Jones informed me that he had examined her two years before, and had then opened the antrum, with a view of determining whether there was any antral disease, but found nothing. I transilluminated the antrum and found nothing abnormal.

Operation was performed on February 1, 1899. The infraorbital and inferior dental were resected as in the preceding case. The portion of the infraorbital measured 2.5 cm. and of the inferior dental 5 cm. in length. After the operation her temperature only once exceeded 99.8° F., and at the end of a week she was entirely well.

Under date of February 28, 1900, she wrote that two months before she had a renewed attack of pain, but not so severe as before the operation. The attack was limited to the upper jaw, the lower one being entirely free. When the pain is severe in the upper jaw she has pain in the right ear, and she said: "The attacks are very easy to bear. I must say I had the best year this last year that I have had for long. I would accept another operation if I should find the need of it again."

Remarks. The history of these cases is the usual history surgically that has been impressed on me by very many operations. The relief is scarcely ever permanent, but at the same time is so great that patients do not hesitate to accept a second operation in view of the great relief they have had from the intense suffering. In spite of only the transitory relief, I believe it is wise to do these peripheral operations until the mortality of Gasserian operations has been materially lessened.[1] For my reasons see a paper by Dr. Spiller and myself in the *Amer. Journ. of Med. Sci.*, November, 1898. These peripheral operations ought to be done early before the disease has had time to invade the ganglion. Very early operations, say after a month or two of ineffectual medication, might even cure permanently.

The nerve is sometimes reproduced with surprising completeness. In two cases I operated on the inferior dental, for Dr. S. Weir Mitchell, on two successive days. After three years I had to operate again on one case, and after six years on the other. In each patient I had removed three small buttons of bone from the lower jaw, 1 cm. in diameter, and chiselled away the intervening bridges of bone. The reproduction of the bone was so perfect that had I not done the operations myself I might even have doubted whether any operation had ever been done. In both I found a complete reproduction of the nerve of a size even larger than that found at the first operation. Both were resected again. The specimens were given to a pathologist, from whom, unfortunately, I never could obtain a report, in spite of the very unusual and interesting character of the specimens. One of the patients has had slight recurrences, but never severe

[1] Amer. Journ. of the Med. Sciences, November, 1898.

enough to require another operation. The other had quite a severe return of pain, which was to some extent relieved by the constant current. Finally she died without ever having required a third operation. In another patient on whom I recently operated for the second time, the first operation having been done by another surgeon, I trephined the inferior maxilla, but found no reproduction of the nerve whatever, though the bone was entirely reproduced.

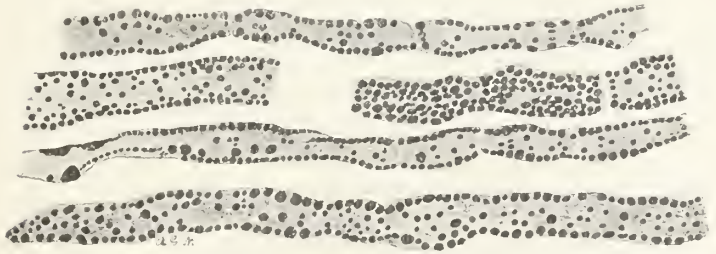
One protest I must make. In nearly every case all the teeth have been extracted. I have never seen the slightest good result from this utterly needless sacrifice.

Dr. Spiller's Report.

Case I.—My report of the nerves resected in this case, with illustrations, was published in a paper by Dr. J. K. Mitchell[1] in 1898, and is reproduced here for the sake of completeness:

"Many, possibly most, of the nerve-fibres of the infraorbital nerve, when separated from one another by teasing and stained by a 1 per

Fig. 2



Degeneration of the medullary sheaths in the form of minute balls stained black by osmic acid.

cent. solution of osmic acid, are found to contain numerous black balls, approximately of the same size. These are nearly equidistant from one another, and are located along the edges of the fibres, leaving, as a rule, the centres free from such accumulations. (Fig. 2.) When the focus of the lens is changed so as to bring other portions of the fibres into view, black balls are apparently found within the centres, but these are probably along the superficial and deep por-

[1] Mitchell, J. K.: Journ. of Nervous and Mental Disease, 1898, p. 400.

tions of the fibres. The medullary sheaths are thus broken into numerous masses of nearly equal size, occupying the normal position of the myelin sheaths. It is exceptional to find masses of degenerated myelin of a size so large as is frequently seen in degenerating fibres. Similar lesions are found in the supraorbital branch of the fifth nerve. Inasmuch as these nerves were taken from the living subject and placed immediately afterward in osmic acid, these myelin balls cannot be regarded as artefacts, or as due to surgical intervention. Such intervention causes a breaking of the fibres into irregular masses, but probably not the fragmentation of the myelin into numerous balls.

"Sections cut with the microtome and stained with carmine and Delafield's hæmatoxylin show more or less round-cell infiltration about the small vessels. The coats of the smallest vessels are not notably thickened, but one vessel of large size, found in some of the sections, presents a thick media and a somewhat proliferated intima. In some of the nerve-fibres pale purple bodies are found which resemble the amyloid bodies and lend some support to the view that the latter are degenerated nerve-fibres. Most of the nerve-fibres contain axis-cylinders, though in some these cannot be seen.

"The hæmatoxylin method of Weigert, used on transverse sections, reveals the presence of the myelin balls within the nerve-fibres in the same manner as the osmic acid shows them in longitudinal sections. They appear as a circle of beads about the axis-cylinder. The nerve-fibres in some bundles appear to be fewer than normal, though this may be due to degeneration of the myelin and therefore imperfectly adaptability to the hæmatoxylin stain."

Case II.—The bloodvessels, even the smallest, in the infraorbital nerve are thickened and the intima is greatly proliferated, considerably reducing the size of the lumen, but the proliferation of tissue is not confined to any one coat of the vessel. The elastic membrane is much thickened, and, in some places, in one of the larger vessels three or more separate elastic membranes are seen. The nerve-fibres, when stained by ammonium-carmine or Weigert's hæmatoxylin, do not appear to be much altered, although occasionally a slightly swollen axone may be seen. In pieces of the nerve teased in the fresh state and stained with osmic acid the disintegration of the

myelin in the form of small black balls is found in a number of fibres, and in some nerve-fibres black balls of a little larger size exist. The description of the infraorbital nerve answers very well for the inferior dental.

This was a case in which the peripheral nerve-fibres were very slightly diseased, although the vessels accompanying the nerves were much thickened.

Case III.—*Infraorbital nerve.* Many of the medullary sheaths are greatly swollen, the axones in a number of nerve-fibres are absent, and these swollen fibres in transverse section appear somewhat granular. In some nerve-fibres the axones are very much tumefied. When stained with Weigert's hæmatoxylin many of the nerve-fibres in transverse sections appear unusually large, and some stain unevenly in shades of black and brown. These large swollen fibres are mingled with small nerve-fibres which stain faintly with the hæmatoxylin. In some of the nerve bundles the nerve-fibres are more nearly normal. The intima of the small vessels is proliferated. No very distinct round-cell infiltration is seen.

Inferior dental. This nerve is less diseased than the infraorbital. The intima in the small vessels is proliferated. The axones are distinctly swollen in a number of nerve-fibres, but the great tumefaction of the medullary sheaths with destruction of the axones is not nearly so common as in the infraorbital nerve. Round-cell infiltration is not prominent.

In two cases of trifacial neuralgia, in one of which resection of the peripheral nerves was done by Dr. John B. Roberts, and in the other by Dr. W. J. Taylor, I have found the black balls of myelin described in the other cases. I have failed to find these black balls of myelin only in one of the cases studied by me, in which resection of the peripheral branches of the fifth nerve was done for the relief of tic douloureux. This was a case of Professor Keen's, in which the pain had existed for twenty years.

I have repeated the description of the nerves in Case I. because of the important findings in this case. When this report was published lesions of this character had not been described. I have since seen this alteration in diseased ulnar nerve-fibres removed after the death of the patient, and this alteration may, therefore, occur in any

diseased peripheral nerve. It is not an artefact, because I have found it in several cases in which the nerves were placed in osmic acid immediately after they were removed from the living subject, and it is not due to any manipulation of the surgeon, because I have found it when the nerve was altered by disease and removed after the patient's death. Professor Obersteiner[1] has referred to my findings, both in print and in a discussion at a meeting of one of the medical societies of Vienna.

A somewhat, but not exactly, similar condition has been described by Elzholz.[2]

Case II. was a very favorable one for a test of the benefit to be derived from early peripheral operations. The nerves were very slightly diseased, and, as Professor Keen has shown, the operation has given relief.

The relief in Case III., in which the nerves were so greatly altered, is also an evidence that resection of peripheral portions of the fifth nerve is a justifiable operation.

Very distinct disturbance of sensation in the distribution of the infraorbital and inferior dental nerves was found ten days after resection of these nerves in Case II., and in my patient, who had formerly been under Dr. Mills' care, and in whom these nerves were resected by Dr. Roberts, the disturbance of sensation after the operation was in the same distribution. Sensation, however, is not always lost after resection of a branch of the fifth nerve, and if lost may soon be recovered, and the recovery is not always due to the regeneration of the nerve. An interesting paper illustrating the truth of this statement has been published by J. K. Mitchell.[3] Quite a number of investigators believe that the facial nerve contains sensory fibres—a view which has been held especially by Frankl Hochwart. Some experimental work has been done to demonstrate the presence of these fibres. I quote from a paragraph in my chapter in *Progressive Medicine*.[4]

"Many writers (van Gehuchten says most writers) now describe

[1] Obersteiner: Jahresbericht über die Leistungen und Fortschritte auf dem Gebiete der Neurologie und Psychiatrie, Bericht über das Jahr., 1898, p. 247.

[2] Elzholz: Jahrb. f. Psych., xvii 3.

[3] Mitchell: l. c., p. 392.

[4] September, 1899, p. 306.

the facial nerve as in part sensory and in part motor. Retzius and, more recently, v. Lenhossék, have shown that the geniculate ganglion is like the cerebro-spinal ganglion, and that the central processes of its cells pass into the nerve of Wrisberg. Van Gehuchten finds, by the method of chromatolysis, that the facial nerve at its exit from the stylomastoid foramen undoubtedly contains a certain number of sensory fibres which arise in the geniculate ganglion. The nerve of Wrisberg is, therefore, the sensory root of the seventh nerve. Amabilino (cited by van Gehuchten) has formed a different opinion. He believes that the peripheral process of the cells of the geniculate ganglion pass into the chorda tympani, and that none of these pass into the peripheral branches of the facial nerve. Van Gehuchten points out that Amabilino found about one-fifth of the cells of the geniculate ganglion normal after section of the chorda tympani, and he very pertinently asks where these cells send their processes if not into the peripheral branches of the facial."

A recent case reported by Biehl[1] is further evidence that the facial nerve contains sensory fibres. A man was stabbed in front of the left ear and had, as a result of the injury, facial palsy with disturbance of sensation in the distribution of the seventh nerve. In this case an involvement of the fifth nerve was improbable.

Pain is not a rare occurrence in facial palsy from exposure to draught, and the explanation usually given is that the fifth nerve has suffered with the seventh. It is probable that in some cases the pain is due to disease of the sensory fibres within the seventh nerve.

Alteration of the peripheral branches of the fifth nerve has been detected in a number of nerves resected for trifacial neuralgia, but the cause of this alteration has not been determined. In two cases I found what appeared to be the explanation of this involvement of the nerve. One of the patients I examined told me that he had fractured his lower jaw, and that after he had entirely recovered from this injury he was attacked with diphtheria, following which pain began in the lower division of the fifth nerve. It seems possible that although the inferior dental nerve had recovered, it offered, as a result of the injury, less resistance to the poison of diphtheria

[1] Biehl: Wiener klin. Woch., Feb. 8, 1900, p. 131.

than the nerve-fibres more commonly attacked in this disease; and, therefore, trifacial neuritis developed. In another of my patients the pain began in the portion of the face which had been the site of erysipelatous inflammation. Here it seemed as though a neuritis had been started by the erysipelas.

It is curious that the right trifacial nerve is more commonly attacked than the left. I noticed the peculiarity in reading the reports of the cases of Professor Keen and others, and at Professor Keen's suggestion Dr. M. B. Tinker has made an examination of the literature to determine the frequency of involvement of the right fifth nerve. In 108 cases in Dr. Tiffany's table, with 24 additional cases collected by Dr. Tinker and the 3 cases reported in this paper by Professor Keen and myself, making 135 cases in all, the affected side is given in 72. In 58 the right fifth nerve was diseased, and in 14 the left.

PATHOLOGIC REPORT ON TWO OF THE GASSERIAN GANGLIA REMOVED BY DR. CUSHING.

By WILLIAM G. SPILLER, M.D.,
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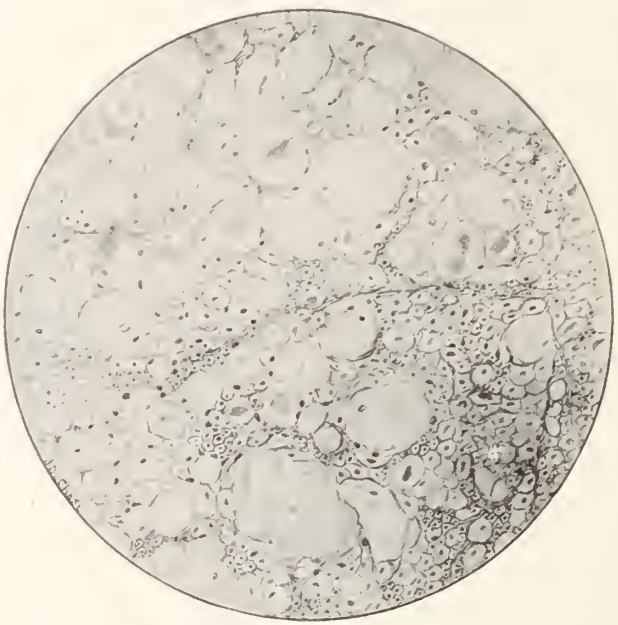
The first ganglion was received hardened in Müller's fluid. Portions of three peripheral divisions were cut separately from the ganglion, and were studied in transverse and longitudinal sections. The body of the ganglion was cut in the longitudinal direction, so that the sensory root and the second and third branches were obtained in the same sections and the relations of the parts could be studied. The stains employed were ammonium-carmine, hæmatoxylin (methods of Weigert and Delafield), acid fuchsin, osmic acid (method of Marchi), and the Rosin stain.

First Division: Weigert's Hæmatoxylin Method. In examining a transverse section in which the decolorization has not been pushed very far and the connective tissue is stained a deep brown—a fair test of the degree of decolorization—one is impressed by the relatively few nerve-fibres which stain a deep black. In each nerve bundle many deeply stained nerve-fibres are found, but the majority of them stain gray or brown. The black fibres are naturally more conspicuous than those faintly stained. This imperfect staining is an indication of alteration in the composition of the medullary sheaths by which the normal chemical reaction with hæmatoxylin is prevented. Not every fibre, even in a normal nerve, stains deeply with hæmatoxylin, possibly because the medullary sheaths of normal nerve-fibres differ in composition, and degeneration and regeneration

are constantly occurring in the same bundle; but such imperfect staining as is seen in this division of the ganglion is probably due to pathologic change. With the Weigert hæmatoxylin method not much swelling of the medullary sheaths can be detected, although some of the sheaths are swollen. When the decolorization is pushed so that perfect differentiation is obtained, chains of black dots, such as I have described elsewhere,[1] are seen in transverse sections of the

FIG. 1.



Oc. 3; Ob. 7. First division of the carmine stain. Many of the axis-cylinders and medullary sheaths are greatly swollen.

medullary sheaths. This is indicative of disintegration of the myelin. Nerve-fibres do not stain evenly in normal sections, but we do not find such a circle of dots in most fibres in normal sections

When the ammonium-carmine and Delafield's hæmatoxylin are employed the axis-cylinders are not colored very deeply. Some of the medullary sheaths are considerably tumefied, but most are of

[1] Spiller (cited by J. K. Mitchell): Journal of Nervous and Mental Disease, 1898.

normal size. (Fig. 1.) Occasionally within a tumefied medullary sheath there is a large and irregular pink mass, the remains of an axis-cylinder, but usually the axis-cylinder cannot be detected within a swollen medullary sheath. Many nerve bundles seem to be perfectly normal, and the diseased fibres are confined to certain bundles.

Fig. 2.

Oc. 3; Ob. 1-12 immer. Swollen and degenerated axis-cylinders within the ganglion (first division).

A much diseased bundle may be found directly in contact with an apparently normal one—possibly there is evidence in this selective tendency of the process for the theory of a cellular origin of the disease. The axis-cylinders are a little more prominent by the Rosin method, and some are distinctly enlarged.

In longitudinal sections of the first division the tumefaction of certain of the medullary sheaths is very evident, and the axis-cylinders are frequently found altered. In some fibres a transverse cleavage of the axis-cylinder has occurred, and in others the axis-cylinder is swollen and of irregular shape. (Fig. 2.) The change in these is not as pronounced as in one of Dr. Keen's[1] cases, studied by me. Often the nerve-fibre has a distinctly fusiform shape, and in the enlarged portion the medullary sheath appears very granular.

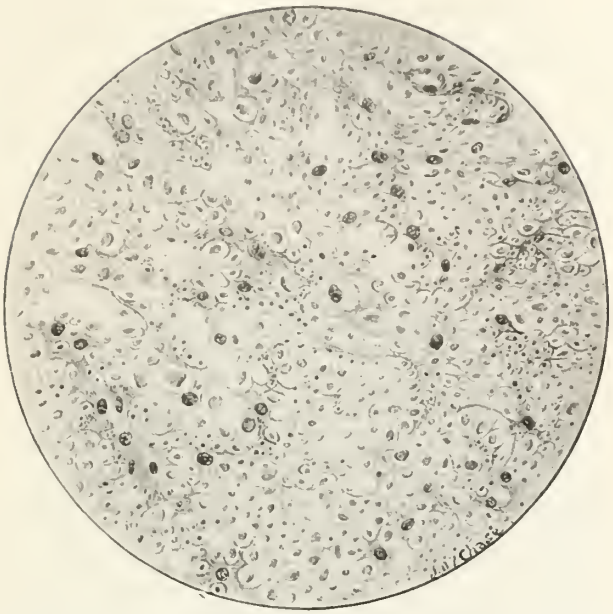
Second Division: Weigert's Hæmatoxylin Method. The nerve-fibres stain more intensely than those in the first division, but in some distinct tumefaction is seen, and the swollen fibres stain in black or light gray or browns, according to the degree of tumefaction, and have rather indistinct outlines when the tumefaction is marked. Some swelling of the medullary sheaths and axis-cylinders is seen by the ammonium-carmine stain. The axis-cylinders possibly take the carmine more deeply than do those of the first division. The bloodvessels do not appear to be greatly altered. The second division, stained by the Marchi method and cut longitudinally, shows a considerable accumulation of black dots in the nerve-fibres. This reaction will be mentioned further on in the description of the body of the ganglion. The tumefaction of medullary sheaths and axis-cylinders is not so evident in longitudinal sections as in those of the first division. In these sections also the vessels appear nearly or entirely normal.

Third Division. The description of the second division answers for the third, and in the latter also the tumefaction of certain medullary sheaths is distinct, and these swollen fibres stain gray with Weigert's hæmatoxylin method. In the employment of the ammonium-carmine a tumefaction of some medullary sheaths with absence of axis-cylinders is seen, and in other parts distinct swellings of the axis-cylinders is evident.

Sensory Root. Many moderately swollen axis-cylinders are found in transverse sections of the sensory root stained with ammonium-carmine. (Fig. 3.) The tissue shows many open spaces, and as the root macroscopically gave evidences in its torn appearance of having

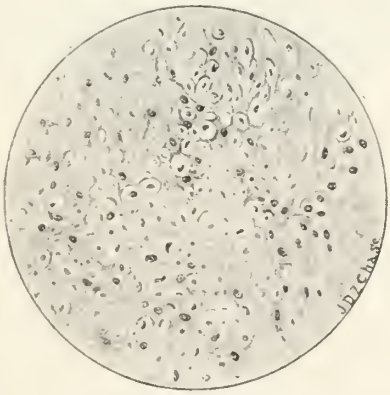
[1] Keen and Spiller: Amer. Journ. of the Med. Sciences, Nov. 1898.

FIG. 3.



Oc. 3 ; Ob. 7 Sensory root by carmine stain. Many of the axis-cylinders are swollen. Compare this degenerated portion of the sensory root with the normal portion of the same root shown in Fig. 4.

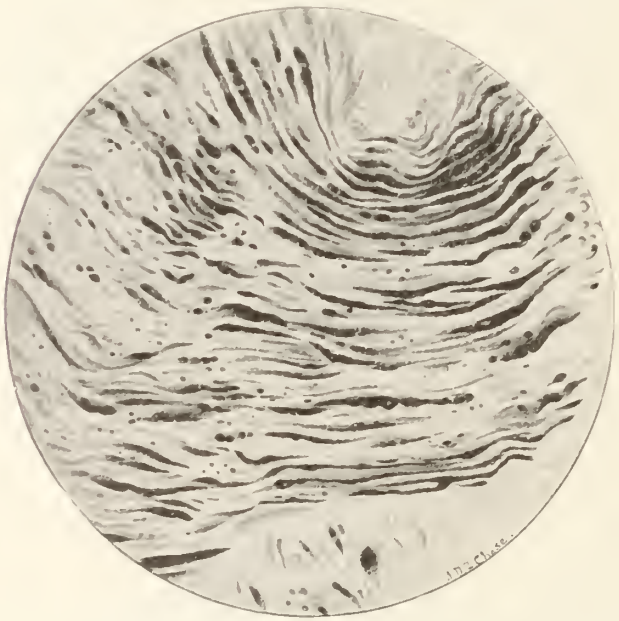
FIG. 4.



Oc. 3 ; Ob. 7. Sensory root, normal portion. This is drawn with the same magnification as is the degenerated portion and should be compared with the latter (Fig. 3). In Fig. 4 a smaller portion of the field has been drawn than in Fig. 3.

been pulled upon, these breaks in the tissue might be regarded as artefacts. The swollen axis-cylinders are found only in this loose tissue, and the tumefaction of axis-cylinders could hardly be produced by surgical manipulation followed by immediate hardening of the tissue. Some of these spaces resemble those that are found when nerve-fibres have degenerated and dropped out, and this loose tissue may be regarded, in part at least, as the result of a pathologic con-

FIG. 5.



Oc. 3; Ob. 5 Degenerated nerve-fibres within the Gasserian ganglion. (Method of Marchi.)

dition. The swelling of the axis-cylinders is not attended by a proportional swelling of the medullary sheaths.

The Ganglion Proper. A space is seen between most of the ganglion cells and the surrounding capsules, and this is evidently due to the hardening process. Some nerve-cells stain deeply with the carmine, others more faintly—a reaction that is seen in normal tissue. The pigmentation of the cells is not excessive, and in almost everyone the nucleus and nucleolus can be seen. The intercellular

tissue can hardly be regarded as increased in amount. The vessels are not strikingly abnormal, and the small vessels have delicate walls. Some tumefaction of axis-cylinders and medullary sheaths is seen in certain parts, but the changes within the ganglion, as studied by the carmine stain, are not very intense. The Marchi method gives most interesting results. The medullary sheaths of many of the fibres passing to the peripheral branches show distinct

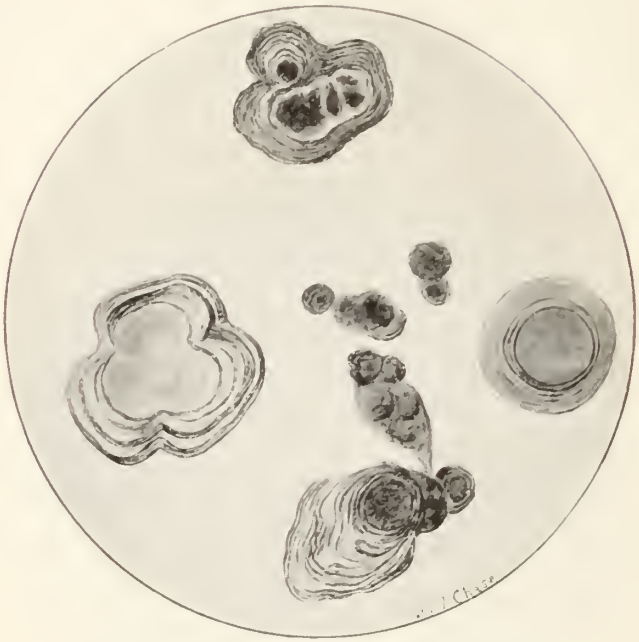
FIG. 6.

Oc. 3; Ob. 5. Sensory root, normal, by Marchi's method.

degeneration (Fig. 5), and a similar degeneration is found within the ganglion. The sensory root presents a marked contrast; it is not entirely free from black dots, but black masses are found in normal nervous tissue in moderate amount when the Marchi stain is used. The sensory root does not exhibit the distinct evidence of degeneration seen in the distal end of the ganglion and the interior parts, and yet one might hesitate to say that this root is absolutely normal. (Fig. 6.) Numerous concentric bodies—brain sand, concentric con-

cretions, etc.—are found within the ganglion, but I have found these in many of the Gasserian ganglia of man that I have studied, and

Fig. 7.



Concentric bodies found in the Gasserian ganglion.

do not regard them as necessarily indicative of disease of the ganglion. (Fig. 7.) The motor root was not obtained.

SUMMARY OF THE PATHOLOGIC CHANGES.

1. Tumefaction of medullary sheaths and axis-cylinders in certain bundles only, in all three divisions of the ganglion. 2. Tumefaction of axis-cylinders in a portion of the sensory root. 3. Numerous spaces in a portion of the sensory root, possibly resulting in part from degeneration of nerve-fibres. The sensory root, therefore, is not normal, and the condition in this root renders a return of the pain possible. 4. Quite intense degeneration by the Marchi method in the centre and peripheral end of the ganglion and its peripheral divisions—a questionable change by this method in the sensory root.

The bloodvessels are not distinctly diseased, and no evidence is offered that the diseased condition of the nerve-fibres resulted from vascular degeneration. Neither do the nerve-cells, by the stains suitable to the method of hardening employed in this case, offer the slightest foundation for the theory of a cellular origin of tic douloureux.

MICROSCOPIC EXAMINATION OF THE SECOND GASSERIAN GANGLION REMOVED BY DR. CUSHING.

A description of this, if given in detail, would be very similar to that of the first ganglion.

First Division. In some bundles the medullary sheaths are much swollen, and here and there a swollen axis-cylinder is found, especially in fibres cut longitudinally. An axis-cylinder may appear to be normal in one part of its course and swollen in another. The greater portion of the first division appears to be normal.

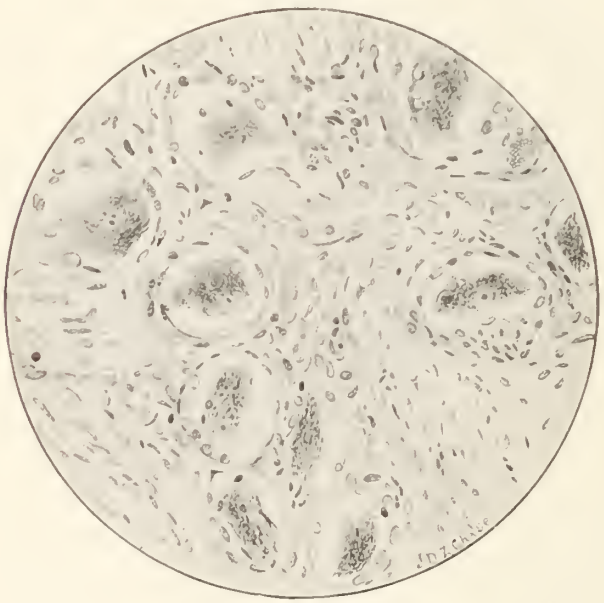
Second Division. The condition of the second division is very similar to that of the first division. Some of the medullary sheaths and axis-cylinders are moderately swollen, but the greater number of nerve-fibres are normal. The small vessels contained within this division of the ganglion have thickened walls. The Marchi stain shows no distinct degeneration. This may partly be due to the hardening in formalin, for while this stain may be employed with formalin-hardened preparations the results are sometimes less successful than with Müller fluid preparations.

Third Division. The third division is more altered than either of the other two; more nerve-fibres are diseased, and the tumefaction of the medullary sheaths and axis-cylinders is greater. No alteration is detectable by the Marchi method.

Ganglion. Most of the nerve-cells appear to be fully or nearly normal, but some are deeply stained and much shrivelled. (Fig. 8.) The cells of the ganglion vary normally in the intensity of their coloring and size, but in the sections of this ganglion the cells which are shrivelled are the ones most deeply stained by thionin. The ganglion was hardened in formalin soon after removal, and these shrivelled cells can hardly be regarded as artefacts. Most of the

cells extend to their capsules without leaving pericellular spaces, and the peripheral portion of the cells contain fewer chromophilic elements than the central portion, and these elements next to this clear peripheral zone are larger and more deeply stained than the others. We know, from v. Lenhossék's description of the cells of the spinal ganglion, that these cells of the Gasserian ganglion are normal. The nucleus is usually very sharply defined and central

FIG. 8.



Oc. 3; Ob 7. Atrophied nerve-cells within the Gasserian ganglion. (Method of v. Lenhossék.)

and the nucleolus deeply stained, but some nerve-cells are greatly shrivelled and deeply stained, and in some the nucleus is peripheral. Some cells are also deeply pigmented.

Sensory Root. The sensory root is normal.

So far as I know the condition of the sensory root of a ganglion removed for trifacial neuralgia has been reported in two cases only—one by Krause and one by myself. Krause found the sensory root diseased and I found it normal. In Krause's case pain returned

on the other side of the face. We cannot say whether or not the occurrence of pain on the side from which the ganglion was not removed was caused by the diseased sensory root of the opposite side. In Dr. Cushing's first case the axis-cylinders of the sensory root were not normal. The tumefaction in these fibres was not excessive, and yet it was sufficient to lead me to believe that a recurrence of the pain in this patient is possible. I have examined the sensory root in three ganglia removed on account of prosopalgia, and only in this one case have I found this root diseased. When we remember that the sensory root of the fifth nerve passes to the sensory nucleus in the tegmentum of the pons, and that many of its fibres descend at the spinal root of the fifth nerve so low as the upper part of the spinal cord, we see that its fibres are distributed over a large area, and that when this root is diseased the diseased fibres are distributed over a considerable extent of the central nervous system. When the Gasserian ganglion is removed the cells in which this root arises are destroyed and the root probably degenerates throughout its length, and it probably loses the power of transmitting any form of irritation after removal of the ganglion. If the root is found diseased at the time of operation, it indicates that the degenerative process has extended centrally beyond the Gasserian ganglion, and the danger of a recurrence of pain is probably greater than when the root remains normal. It occurred to me that possibly the moderate tumefaction of the axones in the sensory root in Dr. Cushing's first case was produced by pulling on the root at the time of removal. The normal condition of the sensory root in the other two cases examined by me hardly permits such a theory. I have taken the sciatic nerve from a cat soon after death, and have forcibly pulled it apart, placed it in Müller's fluid, and examined the axis-cylinders, but I was not able to find the tumefaction of the latter. I fear, therefore, that we must regard the sensory root of Dr. Cushing's first patient as diseased.

In the second ganglion removed by Dr. Cushing the nerve-cells are diseased. This ganglion was placed in formalin and in its preparation the thionin stain of v. Lenhossék could be employed. V. Lenhossék has given such an admirable description of the cells of the spinal ganglion in a man that we know the appearance of cells

of a normal Gasserian ganglion very well. Most of the cells of this second ganglion are normal, but many are intensely shrivelled and deeply stained. It is difficult to believe that these cells are artefacts. The degenerated condition of these cells does not prove that the primary lesions of trifacial neuralgia are in the Gasserian ganglion. The diseased cells are not confined to any one part of the ganglion. Once or twice before I have referred to the investigations of Lugaro and others. These observers found that the cells of the spinal ganglion belonging to the sciatic nerve were much altered when this nerve in the dog was cut, but that no distinct changes in the ganglia were present when the posterior roots or the posterior columns of the cord were cut. Lugaro concluded that the cells of the spinal ganglion are altered in lesions of their peripheral processes, but not in lesions of their central processes. The peripheral process of the nerve-cell in the Gasserian or spinal ganglion has been compared with the dendritic process of the nerve-cell of the spinal cord, and the behavior of the nerve-cell of the ganglion after division of its peripheral process as compared with its behavior after division of its central process indicates that these two processes are of different functional importance. With these facts before us the alteration of many of the nerve-cells in Dr. Cushing's second case is no proof of the primary lesion being in the ganglion. The second division of the nerve had been resected six years previously. Whether the cellular alteration was due to this I cannot say, but it is not probable that it was. In this second case the sensory root was normal. We should naturally expect to find many of the nerve-fibres of this root diseased where many of the cells of the Gasserian ganglion are diseased, but nerve-cells may be more altered than the axis-cylinders arising from them.

A Gasserian ganglion removed on account of trifacial neuralgia and examined by Head,[1] by the Nissl method, was found to be normal, and the nerve-cells so perfect that they could be used as standard specimens of normal staining of the cells of the ganglion. It is a pity that Head does not give the details of this case. With this exception I know of no published report of the examination of

[1] Head, Henry: Albutt's System of Medicine, vol. viii. p. 724 et seq.

the cells of the Gasserian ganglion by Nissl's method in a case of trifacial neuralgia.

In Dr. Cushing's first case the bloodvessels were not distinctly diseased. This would seem to indicate that in this case, at least, the process was not primarily vascular.

Recently two cases have been reported in which the Gasserian ganglion was removed and pain returned on the same side of the face (Friedrich,[1] Garrè[2]), but fortunately these cases are exceptions.

[1] Friedrich, P. L.: Deutsche Zeitsch. f. Chir., 1899, vol. lii.
[2] Garrè: Archiv f. klin. Chir., vol. lix. p. 379.

PROTOCOLS OF MICROSCOPIC EXAMINATION OF SEVERAL GASSERIAN GANGLIA.

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1. *Control from autopsy* 1466 : *Ganglion semilunare sinistrum.* (From a man aged thirty-nine years, dead of chronic interstitial nephritis, arterio-sclerosis, fibrous myocarditis and miliary tuberculosis of both lungs, with right-sided fibrinous pleurisy. No disease of the ganglion was suspected.) Formalin hardening and paraffin embedding were used and serial sections stained by various methods. Nerve-cells are present in normal numbers, the cells larger than the average, generally rounded or oval in shape, and they fill up the capsular spaces. In many of the sections there is a distinct group-like arrangement, several cells being closely crowded together. In some of these groups no capsular cells can be made out around the individual members of the group. The nuclei of the cells are, as a rule, centrally placed. The nucleoli are large and deeply staining; the nucleus itself is often fusiform or spindle-shaped, sometimes round, the alterations in shape being probably due to the fixation. The nucleus in Nissl preparations stains of a light-blue tint. Tigroid is abundant in the cell protoplasm, chiefly in the form of rather minute masses. Some of the cells contain large masses of yellowish so-called "pigment-substance" quite like that seen in the ventral horn cells of the spinal cord. Rather more of the cells show a brownish-black, or almost black, pigmentation, this black pigment being arranged sometimes at the poles of the cell, sometimes in the form of a circle around the nucleus, sometimes in crescentric masses in the cell body. The cells vary in size and shape, large ones predominating. In a few cells the tigroid is scanty about the nucleus, but in the majority it is very evenly distributed throughout the cell body. A number of con-

centric bodies the size of nerve-cells, and some larger, some smaller, are present. They are situated sometimes close to small nerve-cells, apparently crowding them. There is no change in the appearance of the capsular cells. They are not numerous, and are for the most part flat and long-drawn out. The connective tissue about the ganglion shows no alterations. The bloodvessels are not thickened.

2. *Control from autopsy* 1476: *Ganglion semilunare dextrum.* (From a colored woman, aged sixty-five years, dead of epithelioma with multiple visceral metastasis. No disease of the ganglion was suspected.) The nerve-cells vary somewhat in size, but on the whole are tolerably uniform. They are distributed evenly in parts of the ganglion, in other parts they are arranged in definite longitudinal strands of from four to seven cells wide. The cells fill up the capsular spaces. The group-like arrangement met with in the ganglia of autopsy 1466 is not seen here. The nuclei are nearly all centrally placed, and on the whole stain normally, although the nuclear substance takes a slightly bluish tint in Nissl preparations. A number of the cells contain the black pigment mentioned. Very few contain the yellow pigment. The amount of tigroid varies in different cells, some staining much more deeply than others. The tigroid is chiefly present in the form of fine granules. At the periphery of some of the cells there are large bleb-like vacuoles in the cell protoplasm. There are also clear spaces devoid of tigroid at the edges of some of the cells. These may be identical with the *vacuolen* of v. Lenhossék and the disks of Huber. The capsule cells are often irregularly arranged in the periphery, being higher and deeper staining at one or both poles of the cell. In a great many of the cells, however, they are flat and regularly arranged. Only occasionally is there a capsular space not filled up by the cell. A few cells are seen in which there is marked retraction of the nerve-cell body, so that it forms a small crescent at one edge of the capsular space, the rest of this space being either empty or partially filled with a fine granular deposit which looks like coagulated albumin. Scattered through the ganglion are a few foci where small round cells have accumulated. These cells are nearly all small monocular elements of the lymphoid cell type. The majority of them are in the tissue between nerve-cells, but some of them are

found invading the capsule and capsular space of nerve-cells. They may be between the capsule and capsular space of nerve-cells. They may be between the capsular cells and the membrana propriæ on which these rest; occasionally a garland of them is met with between the nerve-cell body and the capsular cells, and sometimes they are actually within the cell bodies of the capsular cells.

This ganglion contains very few of the concentric pale bodies. The connective tissue around the ganglion is not increased in amount. The walls of the bloodvessels show no marked alterations. Along some of the smaller arteries and veins cut longitudinally there is a distinct increase in the number of cells in the tissues just outside. These cells are typical plasma cells of Unna's type.

3. *Control from autopsy* 1480. Man, aged thirty-nine years, who died in status epilepticus following chronic traumatic meningitis; chronic nephritis, œdema of the lungs.

Ganglion semilunare dextrum. No disease of the ganglion was suspected. Nerve-cells are present in normal numbers and distribution, nearly all rather small, very few of the large type being present. There are a good many concentric bodies. Some of them are pale, some of them stain deep, dark blue in hæmatoxylin.

The nuclei are centrally placed. Most of them are round, but some are oval, and a few have jagged edges. The cells are rather pale, the tigroid being scanty. A number of cells show the brownish-black pigmentation; more contain a yellowish pigment. The capsular spaces are uniformly well filled by the nerve-cells. The capsule cells are numerous. An occasional nerve-cell is found far out in the trunk of the nerve. No increase of connective tissue is demonstrable. The bloodvessels are normal. There are no areas of round-cell infiltration or of plasma-cell accumulation.

Ganglion semilunare sinistrum. Sections of this ganglion are similar in appearance to those of its fellow of the opposite side.

4. *Gasserian ganglion from case of cerebro-spinal meningitis.* The ganglion and nerves are swollen and bathed in pus. The latter consists chiefly of polymorphonuclear leucocytes, but there are also many small monocular leucocytes and a number of larger phagocytic cells containing several of the smaller cells within their protoplasm. The nerve-cells are present in normal numbers. The actual single

nerve bundles and masses of ganglion cells are scarcely invaded by the purulent process, the pus lying between the individual nerve bundles and at the periphery of the various ganglion cells and masses. The cells stain intensely in Nissl preparations, and the tigroid is abundant. The capsular spaces are well filled. The nuclei, as a rule, are centrally placed, and they are distinctly swollen. The nucleoli are present and sometimes swollen; no evidence of general chromatolysis can be made out, the most marked alteration being the extreme swelling of the nucleus. Sometimes the nucleus is so swollen as to fill up most of the cell, leaving a rim of deeply staining protoplasm peripheral to it. In one mass of ganglion cells thus altered the nuclei are eccentrically placed, and in some of the cells the tigroid has almost disappeared. It is probable that the nerve-fibres connected with these cells have been injured in the inflammatory process. The blood in the bloodvessels shows a slight leucocytosis; the bloodvessels show no demonstrable alterations.

5. *Experiment on a dog's Gasserian ganglion.* (Operation by Dr. H. W. Cushing, with evulsion of the N. maxillaris on the right side.) The right and left semilunar ganglion were fixed in alcohol at the end of fifteen days, with paraffin embedding and staining by Nissl's method. In the ganglion on the right side are rows of ganglion cells chiefly situated in the middle of the ganglion, which show the typical "reaction at a distance;" that is, displacement of the nucleus to the side of the cell with chromatolysis. Where the changes are most intense nearly all the cells have undergone alteration. At the peripheries of this intense area altered cells are scattered in among healthy cells.

6. *Ganglion semilunare dextrum in case of tic douloureux.* (Removed by operation by Dr. H. W. Cushing, January 15, 1900.) The ganglion has been removed entire along with the stumps of the N. ophthalmicus, N. maxillaris, N. mandibularis and N. trigeminus. In Marchi preparations of the N. trigeminus a number of the fibres are markedly swollen and show diffuse fine black droplets. Similar changes are found in the N. ophthalmicus and N. maxillaris, though not to so marked a degree. There is, however, no outspoken typical Marchi degeneration in any of the nerves. The nerve-cells in the ganglion do not appear to be diminished in number; the size of the

cells varies; there are rather more of the very large ones than normal. A great many of the cells are irregularly retracted into diffusely staining chromophile masses, leaving wide capsular spaces. Some of the cells not contracted are paler than normal, having less tigroid in the Nissl preparations. Still, a good many of the cells appear to have the normal amount of trigoid. The peripheries of the cells are irregular. The nuclei are, as a rule, centrally placed, round and contain normal-looking nucleoli. Yellow pigmented cells are abundant. Black pigmented ones are far less numerous. Concentric bodies are abundant in this ganglion, some of them being very large. The changes in this ganglion are very much less marked than in the next case. There is no increase of connective tissue. No vascular changes are to be made out, and no accumulations of foreign cells in the ganglion. The capsular cells are for the most part low and flat.

7. *Ganglion semilunare sinistrum in case of tic douloureux.* (Removed by operation, by Dr. H. W. Cushing, January 17, 1900. Surgical number, 2907). The ganglion was cut into serial sections, some of them being stained by Nissl method; others by hæmatoxylin and eosin, others by Van Gieson's method. The ganglion has been removed *in toto,* but is somewhat lacerated. The nerve-cells are generally normal in size, though some of the cells look swollen. A large number of the cells are irregularly retracted, leaving a wide capsular space, portions of the cell protoplasm sticking out like prongs to the periphery. The cells stain very irregularly; a large proportion of the cells are chromophile in the sense of Nissl, that is to say, they stain, by Nissl's method, a deep, dark-blue color, so that it is very difficult to make out anything of their internal structure. The nucleus cannot always be seen, owing to the dark staining, but sometimes it can be made out, and then is usually centrally placed. Throughout the deeply staining blue substance, pale, vacuole-like areas can be made out, and masses of yellow pigment are also present in these chromophile cells. Of the cells which are not chromophile, a large proportion are very pale, almost devoid of tigroid, the little that is present being situated at the peripheries of very small pale areas scattered through the ground substance of the cell body. The nuclei of these cells vary in size and shape; some of the nuclei look

swollen; others look contracted. Nucleoli are frequently absent from the cells, although in many they are present and are normal in size and position. In a few the nucleolus is seen outside the nucleus, lodged in the cell protoplasm. In some there has been fragmentation of the nucleus, two and three pieces of the latter remaining side by side in the cell body. The pale cells in hæmatoxylin and eosin preparations show a delicate network-like appearance stained in hæmatoxylin. Scarcely any normal-looking cells exist. Cells with black pigment and with yellow pigment are present, the latter being more abundant than the former.

There are a few concentric bodies. There is no increase of connective tissues about the ganglion demonstrable. The small blood-vessels in the ganglion are dilated; there is no round-cell infiltration.

OPERATION IN TIC DOULOUREUX.

A few convictions with regard to operative measures in tic douloureux may here find expression:

1. If a ganglion be entirely removed there need be no fear of a return of pain from irritation of the stump of the N. trigeminus left behind, for all of the axons of this stump will degenerate to their terminations in the pons and medulla, down as far as the cervical cord. The end of a nerve in an amputation stump is not analogous.

2. Complete removal of a Gasserian ganglion abolishes the possibility of calling forth sensations in consciousness by applying stimuli to the domain of peripheral distribution of the nerves connected with the ganglion of the corresponding side; retention of sensation on peripheral stimulation after operation indicates incomplete removal.

3. If pain persists, paroxysmally or continuously, after complete removal of the ganglion, or after evulsion of the N. trigeminus from the pons, the ganglion being left *in situ*, a lesion of the central neurons—of the second or of higher orders—of the trigeminal afferent conduction path is indicated.

4. In *tic douloureux* due to disease of the peripheral set of trigeminal sensory neurons, relief should be as complete and permanent by cutting the N. trigeminus between the ganglion and the pons and evulsing the central end without removal of the ganglion as when the ganglion itself is excised.

NATURAL HISTORY OF TIC DOULOUREUX, WITH REMARKS ON TREATMENT.

By CHARLES L. DANA, M.D.,
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[Read at a special meeting held April 20, 1900.]

If we hope to judge wisely as to how much good certain forms of treatment do for tic douloureux, we must know its natural history, which is not yet very fully understood. For though the disease is common, its origin is often unexplainable, and there is none in which so much desperate effort is made to break up the course of the attacks and interfere with the natural symptoms. I have thought, therefore, that I could serve the purposes of this discussion best by devoting a good part of my time to this side of the subject.

It is one of the advantages of somewhat mature years that we are able to take a wider view of the course and of the history of the diseases as shown in our patients, and I have been able to make a close study of a number of my own cases from the beginning to the end. What I have to say now is based very largely on the recorded history of over fifty cases of tic douloureux, some of which I have had under observation for from five to ten years, and most of which I know the entire history of from the outset.

Pathogenesis. Tic douloureux is, as I understand it, a degenerative neuralgia occurring in the great majority of cases, at or after the middle period of life and due to a degenerative change, sometimes amounting to neuritis, in the nerve and its ganglion, and probably in the bloodvessels which supply it. It attacks women[1] about twice

[1] As to sex, among 57 cases there were: males, 20; females, 37. Under 35 there were: males, 3; females, 10. Over 35: males, 17; females, 27. Ages at onset of disease: 20 to 30, 5; 31 to 40, 7; 41 to 50, 7; 51 to 60, 17; 61 to 70, 15; 71 to 80, 5; 81 to 90, 1. Side affected: right, 22; left, 14.

as often as men, taking the whole period of the disease, but it rarely attacks men before the age of forty. I have seen only three cases of this kind of tic douloureux in a man under that age, whereas there have been ten women in whom the disease began under that time. Thus, it may be said to be at least three or four times as frequent in women under forty as it is in men.

There is rarely any distinct hereditary taint, still I have had one patient, aged forty-six, whose father suffered from the disease at the same time. In another patient the grandmother had tic during her old age, and in a third the mother also had it. The most common neurosis found in the family is that of migraine, but this is so common generally that it can hardly be said to count in the personal history.

The most common previous nervous disorder in the patient, however, is also that of migraine, which I find in one-fourth of my patients. A common exciting cause is some overwork or strain (14 per cent.).

No doubt many have observed that the initial disease begins as an acute trigeminal neuritis associated with herpes. This was the case in six of my patients. This fact of an initial herpetic neuritis is one of much importance as pointing to the origin of the disease in some cases from an infection causing a distinct peripheral neuritis. In a number we find that the cause is a local disease of the gums or teeth. In about 20 per cent. then there is an initial neuritis or local disease. I have also seen it start as the result of la grippe in three cases, and of exhausting hemorrhage in another.

Tic douloureux is, in the vast majority of instances, a disease of the second or third branch of the trigeminal nerve. It is extremely rare for it to begin in the ophthalmic, or remain localized in that branch alone, whereas it often affects the second and third branch alone for a long period of time. Among forty-five chronic cases I find only one instance in which the trouble was confined alone to the first or upper branch of the fifth nerve.

Types. Cases which begin in women in early life have always been a puzzle to me, and I have always been doubtful as to their true character, although they do eventually seem to assume the type of a true tic. In carefully studying my own histories, it seems to

me that these early ones are to be explained in this way: The patients are almost always young women suffering from migraine, and their trouble is preceded by frequent attacks of this malady. After a number of years this migrainous disorder seems to become chronic and in a measure localized. It then settles in the second or third, or perhaps all three branches of the nerve. Such patients have, however, in addition to the sharp pains along the course of the nerve, diffused pains involving the head and often each side of the face. Cases that develop at the degenerative period of life are rarely associated directly with migraine, although one often gets a history of some migrainous trigeminal neuralgia, or attacks of migraine occurring early in life. The malady, however, often seems to settle down on the nerve as the result of the exciting causes already referred to, although referring to no particular predisposing neurosis.

I am disposed, in view of the facts referred to, to think that there are at least two particular and distinct types of tic douloureux, not including the purely symptomatic pains due to extraneural disease.

The first I should call the migrainous type of early life, and I consider it an evolution of a definite trigeminal neuralgia on the basis of a migrainous neurosis. It is seen oftener in women. The trouble is originally central and not a disease of the Gasserian ganglion. Later it is possible that the fibres of the trigeminal nerve, through some inherent defect, incur degenerative changes and a true degenerative neuralgia develops. But it is to be borne in mind that in the early stages at least there is very little real disease in the fifth nerve. It is a disorder of the central sensory neuron.

The second type is the more common one, occurring after the age of forty and affecting men at least half as often as it does women. Here the disease sometimes starts as a true infective neuritis in the form of a herpetic or ascending neuritis, due to some local disease of the gums or the antrum, or the osseous tissues. Some of these herpetic forms run an acute course, lasting only a few weeks or months, and are easily cured.

Tic douloureux is sometimes purely symptomatic of local disease of the bone, of a tumor, etc.

Natural Course. The natural course of these different forms of neuralgia is somewhat as follows: The persons who suffer from

migrainous tic in early life, finally localizing itself as a true tic, rarely get cured, but have a long and tedious period of suffering. The prognosis is more unfavorable than that of ordinary migraine; however, like the latter, under exceptionally good constitutional conditions, periods of long remission occur, and sometimes the trouble spontaneously disappears. This tendency to disappear is probably somewhat increased after the age of fifty, just as is the case with migraine, and I have a patient in whom the whole trouble disappeared at this age, after she had had a tic for twenty-nine years, the cause of the cure being asserted to be "Christian Science."

These cases also of the migrainous type, so far as my experience goes, are made worse by the ordinary operations for tic. I do not know what the result of the removal of the Gasserian ganglion would be in such early cases, but I have never seen any cures from the other minor surgical operations, although I have had a number of patients who have had these made on them at different times. Such operations may give temporary relief, but not always. Patients of this class are ill adapted for operation, because the trouble does not lie exclusively in the trigeminal nerve, but also in the central nervous system.

Of the second class of patients the natural history is more definitely known. Among seventeen cases of which I have definite histories from beginning to end, in seven the disease has stopped or has been cured. In six others it has been improved and kept under control, while in four its progress has not been affected by anything that has so far been done. Thus it appears that in at least 20 per cent. the disease is amenable to treatment or is relatively benign.

In these cases in which the trouble has been relieved entirely for a period sufficiently long to feel that it is practically under control, one attributes this relief to animal magnetism, two to operations, three to the minor operations of removing the tooth and washing out the antrum, one to "Christian Science," and the remainder to various measures, mostly the use of strychnine, tonics and rest or the natural course of the disease. With two exceptions all the cases which were relieved or cured occurred in persons in whom the trouble began after the age of forty, and in whom it had been lasting for not more than eight years, the average being about four.

The average duration of the disease, in which the malady stopped or was brought under control without any very definite or special medical treatment, was about five or six years, and my conclusion is that tic douloureux, beginning in persons at the age of forty or over, has a natural tendency to run its course, this course ranging from five to twelve years. It usually reaches its height at the fifth or sixth year, and then has a tendency in some cases to spontaneous cure. The duration[1] of non-curable forms may be even the life of the patient. I have seen cases in which the duration was twenty-nine and thirty-two years, others where it lasted twelve, seventeen, and eleven.

One of the most characteristic features in the natural history of tic douloureux is its tendency to remissions. These occur for periods of one or two months, but they can be artificially produced by various methods of treatment. The surgical treatment will usually produce a remission of one-quarter to one-half a year; the medical treatment will often produce remissions of from six to ten months. These can be renewed by further systemic therapeutics. After the disease is long established, however—*i. e.*, after six or seven years, there is apparently no measure, except a surgical one, which will absolutely and surely stop it. The surgical operations, except those for the removal of the Gasserian ganglion, produce only temporary remissions: all the medical measures have the same effect. As to the relative efficacy of the two measures, I can say, without any bias, that in most cases we can secure a remission by the medical measures almost as certainly as we can by surgery, except by the major kind. No drugs, however, and no surgical operation will arrest the disorder permanently. They will only lengthen the remissions and perhaps shorten the course.

It is perhaps known that for some years I have advocated a systemic treatment for tic douloureux by means of heroic doses of strychnine. After experimenting with this method of treatment for now over six or seven years, and including about fifteen cases, I have reached this conclusion regarding it: In early cases of this

[1] Duration of cases: cured or well-controlled without surgical measures—At 55 to 60, 5 years; 64 to 72, 8; 55 to 59, 4; 66 to 69, 3; 61 to 64, 3; 21 to 50, 29; 60 to 66, 5; 57 to 61, 4; 58 to 61, 3; 58 to 63, 5; 61 to 71, 10; 82 to 88, 6. Side affected: right, 22; left, 14.

kind, that is to say, in the first and second years, the strychnine treatment will almost invariably arrest or control the disease in anæmic and exhausted patients. In cases that have lasted over six or seven years, and in those with neuritis and selerosis, the result is doubtful, and there may be a failure to secure even a remission. In anæmic ones, however, even of long standing, it is often more effective, though relapses will occur. Among fifteen cases I have had only four distinct failures. In the very old cases, lasting fifteen or twenty years, medical treatment is practically valueless, no matter what is done.[1]

I have few favorable statistics to report of the results of surgical operations. I have seen absolutely none in which these succeeded in curing the patient, although one of my cases included that of a patient in whom the Gasserian ganglion was removed. I have seen operations, however, produce remissions lasting for two years. I have, as already stated, had three patients in whom the trouble ceased after the tooth had been removed and the antrum washed out. In no case have I known relief by the removal of teeth, or at least nothing more than temporary relief. Among 42 cases, 11 had had 24 operations, with no permanent cure in any case. One only was that of removal of the Gasserian ganglion.

As to the general matter of treatment it is better to record experiences than opinions. I have seen one case apparently helped by the opium treatment; but it is dangerous and not usually effective. Three of my patients had become opium habitues. Three patients, after long courses of medical treatment, and in one instance after an unsuccessful operation, were cured by removal of a tooth and washing out the antrum. In another this did no good. One patient thinks she was cured by animal magnetism, and one by "Christian Science." Remission followed in one case after prolonged use of the static breeze an hour daily for two months. This I believe was purely a natural remission. One patient had her ovaries removed, with the result that the face was worse and she had a vaginal neuralgia.

[1] The details of the technique of this method are given in full in the Post-Graduate, July, 1896, and in the 4th edition of my Text-book of Nervous Diseases.

I have seen little good result from aconite or from any of the benumbing or narcotic drugs. Medical treatment is most successful which is addressed to the arthritic state, when that exists, and to the arterial sclerosis and gouty taint. In some of this latter, men of full habit and hard arteries, tonic measures do harm, and after a course of rest and arterio-sclerotic or neuritic measures, surgery should be promptly applied. All measures will be more effective if applied at the beginning of the disease, and again at the fourth or fifth year when the trouble is at its height; or again at the period of the climacteric in those whose neuralgia began relatively early in life.

In conclusion, therefore, I should say that the early forms of tic douloureux, such as I have called a "migrainous tic," occurring usually in women, should not be operated on. There are some exceptions to this, however, in which tic douloureux occurs in early life, due to a distinct local disease, such as an inflammation of the nerve, or of the antrum, or of the jaws. In true tic of the degenerative period of life, prompt medical treatment will usually control the disease and operation is rarely indicated at first. In tic which has lasted three or more years, it may be safely said to the patient that medical treatment may produce a remission, and that this remission may be repeated, and that eventually the disease may be controlled by repeated treatment, but this is not at all sure. It may be said here, too, that a minor operation may give more relief than medical treatment. The question of prescribing major operations must be decided in each individual case, on its special merits.

TABLE SHOWING DURATION AND TREATMENT OF FORTY-TWO CASES.

1. Male. Duration, 55 to 60; spontaneous cure.

2. Male. Duration, 64 to 72; strychnine; relieved; 8 months relapse; cured for 3 years by removal of tooth and washing antrum.

3. Female. Duration, 55 to 59; 3 operations; no relief; strychnine; remission 6 months; strychnine; remission 8 months; cured—animal magnetism 10 months.

4. Female. Duration, 66 to 69; strychnine; improved—fairly well 2 years.

5. Male. Duration, 61 to 64; strychnine; relieved 1 year; slight remission; relieved 2 years.

6. Female. Duration, 21 to 50; strychnine; no result; cured by washing out antrum and "Christian Science."

7. Female. Duration, 60 to 66; strychnine; relieved; remission; cured by washing out antrum; patient took morphine.

8. Female. Duration, 57 to 61; strychnine; relieved; disease controlled 2 years.

9. Male. Duration, 22 to 31; operation; relieved for 2 years; return.

10. Male. Duration, 31 to 33; strychnine; total failure; operation; no return in 1 year.

11. Female. Duration, 58 to 63; strychnine; relieved; disease controlled 2 years.

12. Female. Duration, 68 to 71; strychnine; relieved 6 months; remission; relieved again; disease still under control.

13. Female. Duration, 52 to 58; strychnine; relieved for 1 year; strychnine; no relief; patient lost sight of.

14. Female. Duration, 55 to 57; strychnine; relieved for 8 months; relapse?

15. Female. Duration, 31 to 47; strychnine; slight relief.

16. Male. Duration, 31 to 35; static electricity; remission 2 months; static electricity; remission 6 months; static electricity; remission 2 months; operation; no relief; strychnine; remission 4 months; strychnine; remission 4 months; return.

17. Male. Duration, 61 to 63; strychnine; no relief; disease still continues.

18. Female. Duration, 25 to 65; 3 operations; no relief; strychnine; no relief.

19. Female. Duration, 30 to 44; 2 operations; no relief.

20. Female. Duration, 45 to 52; strychnine imperfectly given; no relief; disease disappeared; patient suffered from melancholia.

21. Male. Duration, 50 to 62; operation; no relief; 3 other operations; no relief.

22. Male. Duration, 31 to 40; no regular treatment; patient somewhat better.

23. Female. Duration, 65 to 72; 2 operations; no relief; strychnine; no relief.

24. Female. Duration, 24 to 42; not heard from.

25. Male. Duration, 46 to 48; strychnine; somewhat improved.

26. Female. Duration, 25 to 28; relieved without any special treatment.

27. Female. Duration, 45 to 52; morphine.

28. Female. Duration, 30 to 40; nasal pain.

29. Male. Duration, 58 to 67.

30. Male. Operation; relapse.

31. Female. Duration, 82 to 88; 2 operations; relief 4 months; opera-

tion; no relief; medical treatment 6 months; pain ceased; patient died 4 months later of old age.

32. Male. Duration, 33 to 43; strychnine; relief.

33. Male. Duration, 51 to 54; strychnine.

34. Male. Duration, 50 to 60; strychnine; relieved.

35. Male. Duration, 39 to 47; operation; relieved 1 year; strychnine; improved.

36. Female. Duration, 66 to 68; strychnine; relief,

37. Female. Duration, 60 to 65; general medical treatment; relief; lost sight of.

38. Female. Duration, 53 to 56; medical treatment; relief.

39. Female. Duration, 55 to 60; medical treatment; lost sight of.

40. Male. Duration, 55 to 61.

41. Female. Duration, 71 to 75.

42. Female. Duration, 23 to 35.

A METHOD OF TOTAL EXTIRPATION OF THE GASSERIAN GANGLION FOR TRIGEMINAL NEURALGIA.

BY A ROUTE THROUGH THE TEMPORAL FOSSA AND BENEATH THE MIDDLE MENINGEAL ARTERY.

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[Read at a special meeting held April 20, 1900.]

Of the many diseases which, on therapeutic grounds, are supposed to occupy a border-line position between the provinces of the physician and the surgeon, perhaps no one more than intractable epileptiform neuralgia illustrates so well the dictum of that renowned Philadelphian and friend of many doctors, Benjamin Franklin, to the effect that "He is the best physician who knows the worthlessness of the most medicines."

Granting the premise in all cases of true tic douloureux, the neuralgia quinti major of Henry Head, in which all three divisions of the trigeminal nerve are affected, that surgical measures alone can with any degree of certainty be depended on to afford relief from this horrible affliction and that the removal of the Gasserian ganglion must ultimately be contemplated, it is to be regretted that this final procedure should generally be regarded as one hazardous in its performance and uncertain in its permanent effects. Two factors may be held responsible for the ill-repute in which the ganglion operation at present stands; in the first place the considerable attendant mortality, ordinarily placed at 20 per cent.; and, secondly, the impression which is prevalent regarding the possibility of recurrence of the neuralgia, an impression which has been occasioned by the reports of cases in which incomplete operations have been performed with a subsequent return of pain.

Almost without exception descriptions of the operation relate in appalling fashion the severity of the hemorrhage which has ensued during one step or another of the procedure, and which has in most instances precluded the possibility of a total extirpation. Granting an equal familiarity with the surgico-anatomic relations of the ganglion semilunare, especially those referable to its dural envelope, the successful accomplishment of its removal, whatever the method employed, will depend entirely on the degree with which an operator may avoid a bloody wound, since a clean, dry field is almost a *sine qua non* for the manipulations, which even on the cadaver offer considerable difficulty. The objective point of the operation is necessarily located at the bottom of a close-walled operative well whose depth varies from 5 to 8 cm., and blood-staining even in comparatively slight amount will obscure the ganglion and be incompatible with its complete removal. Consequently, under circumstances which have demanded that the operation be conducted in two or three sittings with periods of tamponade of from two to five days, with possible preliminary ligation of the carotid, and almost without exception leaving wounds which have required drainage, there is little occasion for surprise that many operators have contented themselves with division of the second and third branches—N. maxillaris and N. mandibularis—and random removal of the adjoining portion of the ganglion with the aid of blunt hooks or the curette. It has been a not infrequent experience at the hands of those who have been careful enough to submit to histologic examination the tissues removed, under such circumstances, from the supposed site of the ganglion, to find that no ganglionic structure whatever was demonstrable, and that recurrences should be recorded in such instances is no occasion for disparagement of the operation.

Apparently, heretofore in but few cases, notably by Keen[1] and Krause,[2] in Coelho's[3] case, and in a few others, has the ganglion been removed *in toto* and as a recognizable entity worthy of his-

[1] Keen, W. W., and W. G. Spiller: Remarks on Resection of the Gasserian Ganglion, with a Pathological Report on Seven Ganglia. Amer. Journ. Med. Sci., vol. cxvi. (1898) p. 503.

[2] Krause, Fedar: Die Neuralgia des Trigeminus nebst der Anatomie und Physiologie des Nerven. Leipzig, Verlag von F. C. W. Vogel, 1896.

[3] Coelho, Sabino: Ablation du ganglion de Gasser avec arrachement protubérantiel du trijumeau dans un cas de névralgie faciale rebelle. Revue de Chir., Mai, 1899, T. xix. p. 623.

tologic study. Relative to photographs of the ganglia removed by Krause and Doyen,[1] Marchant[2] has commented on the necessity of microscopic examination of the tissues removed in confirmation of their presumed ganglionic character. The experience of finding no ganglionic elements in such material has occurred even to operators as skilful as Dr. Keen.[3]

Whether after complete extirpation of the ganglion a continuance or recurrence of painful stimuli of the central system of neurons may follow, must remain a matter of temporary uncertainty, since observations on such conditions have only held over a period of very few years. It is certain, however, from physiologic knowledge of the process of nerve repair, that there can be no peripheral regeneration of the lower system of sensory neurons after the ganglion has been removed, such regeneration as always occurs after division or evulsion of the individual roots, and possibly after incomplete removal of the ganglion itself. Experimental evidence[4] in the case of the spinal cord goes to show that division of the central axons of peripheral sensory neurons does not preclude the possibility of physiologic regeneration between a spinal ganglion and the cord. This same principle is applicable to the cerebral sensory ganglia, and in consequence by analogy with the spinal cord it is evident that the simple division of the sensory root of the fifth nerve—N. trigeminus—which procedure has been proposed as an alternative for the removal of the ganglion itself, would be inefficient. The degenerative changes found by Dr. Spiller in the sensory root of one of my cases has aroused a suspicion of possible recurrence of the pain; in this

[1] Doyen, E.: L'extirpation du Ganglion de Gasser. Archives provinciales de Chirurgie, Juli, 1895, T. iv. p. 429.

[2] Marchant, Gérard et Heuri Herbert: De la résaetion du ganglion de Gasser dans les névralgies faciales rebelles. Revue de Chirurgie, 1897, T. xvii. p. 287. These writers say in a foot-note, p. 295: "Nous aimerions malgré tout, voir figurer, dans ees observations, a eoté de la photographie, le résultat de l'examen histologique. Un ganglion arraehé, tordu ou broyé par les mors de la pinee est en général assez altéré dans sa forme et peut donner lieu a de eertaines illusions. Nous ne pouvons nous empêeher de penser que dans un cas (Dennetiers: Soeiete de Chirurgie, 15 Juillet, 1896), ou, d'après son aspeet, la partie enlevée resemblait tant bien que mal a un gauglion, l'exemen histologique le plus conseieneieux n'a pu déeeler la présence d'aueune eellule ganglionnaire. Il en fut de méme daus deux cas de Keen."

[3] Keen, W. W.: Remarks on Operations on the Gasserian Ganglion, with the report of five additional cases. Amer. Journ. of the Med. Sei., Jan. 1896, vol. exi. p. 59. Case II.

[4] Baer, Dawson and Marshall; Regeneratiou of the Dorsal Root Fibres of the Second Cervical Nerve within the Spinal Cord. Journ. of Exp. Med., Baltimore, 1899, vol. iii., No. 1.

case, however, one in which there was a return of neuralgia a few weeks after two earlier peripheral operations, there has been no sensation of pain whatever since the operation nine months ago. (See Case I.)

In view of the foregoing data, I believe that it may with propriety be stated:

1. That the probability of non-recurrence bears a direct relation to the degree of entirety with which the ganglion has been removed.

2. That the satisfactoriness of the operation is commensurate with the degree of preservation of the ganglion during its removal and the consequent possibilities of a histologic identification of its elements.

3. That the evolution of the operation must be in the direction of avoidance of hemorrhage which will interfere with the manipulations necessary to successfully liberate the ganglion.

OPERATIVE METHODS IN GENERAL.

A variety of methods more or less familiar have been proposed by means of which the ganglion may be approached, the two most widely quoted being associated with the names of Hartley and Krouse and of William Rose. The fact that Rose, in his original operation,[1] excised the superior maxilla in order to reach the ganglion only emphasizes the terrible nature of the malady which made justifiable such a mutilating procedure undertaken for its relief. In the subsequent development of the operative method by what is known as the *pterygoid route*, which Rose[2] and his followers have adopted, the ganglion is approached from below by a trephine opening at the roof of the zygomatic fossa. If for no other reason than that the ganglion can hardly be removed *in toto* from such a situation, this route should be abandoned, and its uncertainties are evidenced by descriptions of methods by which this structure may be broken up with a curette and thus destroyed.

[1] Rose, Wm.: Removal of the Gasserian Ganglion for Severe Neuralgia. The Lancet Nov. 1, 1890, vol. ii. p. 914.

[2] Rose, Wm.: The Lettsonian Lectures on the Surgical Treatment of Trigeminal Neuralgia, Lecture II. The Lancet, Feb. 6, 1892, vol. i. p. 295.

The methods proposed by Doyen,[1] Quénu,[2] Poirier[3] and other French surgeons with an approach by what is known as a combined *temporo-sphenoidal route*, also possess some of the disadvantages of the method of Rose, for though the exposure is better, the ganglion is approached from below through the bloody area of the pterygoid plexus, with necessary ligation of the internal maxillary artery, and after location of the inferior maxillary nerve—N. mandibularis—the roof of the zygomatic fossa is rongnered away to the foramen ovale and an attempt made to remove the ganglion by using this nerve as a guide to its position. Personal experience with this operative method, though limited to the cadaver, has demonstrated that it is exceedingly difficult to remove the ganglion from this situation in a satisfactory degree of preservation, and furthermore, as will be seen, the blood-supply to the ganglion is almost entirely from below and is especially abundant in the neighborhood of the foramen ovale, consequently the difficulties of this method during life must be considerable. Jacob[4] has recently described an operation in which the infraorbital branch—N. infraorbitalis—of the superior maxillary nerve is first located at the floor of the orbit, the skull trephined and the ganglion approached and identified by means of this nerve in much the same way that the inferior maxillary nerve is utilized in the last-described procedure, but inasmuch as these nerves are accessible and plainly recognizable within the skull, it seems unnecessary to demand any such preliminary extracranial sign-post to the seat of operation.

The fact, however, that French surgeons since Doyen have clung to the temporosphenoidal route, gives evidence that its possibilities are deserving of consideration; but inasmuch as the great majority of operators have followed the lead of Hartley[5] and of Krause[6] by

[1] On Extirpation of the Gasserian Ganglion. Ref. Annals of Surgery, 1896, vol. xxiii. p. 69.

[2] Quénu et Sibileau: Bull. Ac. Med., 10 Jan. 1894; indorsed by Tichonowitch, Centralblatt für Chir., 24 Marz, 1900, S. 322.

[3] Poirier, P.: Resection du Ganglion de Gasser; arrachement protubérantiel du trijumeau. Gaz. d. Hôp., Paris, 1896, T. lxix. p. 808–810

[4] Jacob, O.: Un procédé de resection du Ganglion de Gasser. Revue de Chir., 1899, T. xix. p. 29.

[5] Hartley, Frank: New York Med. Journ., March 19, 1892. Subsequently—Intracranial Neurectomy of the Fifth Nerve. Annals of Surgery, 1893, vol. xvii. p. 511.

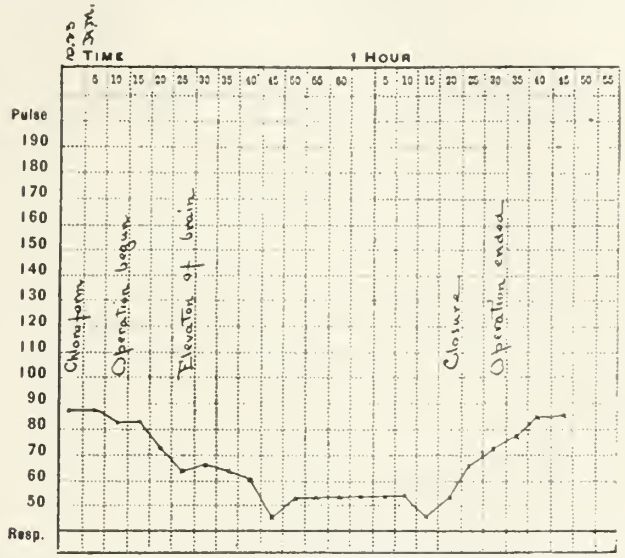
[6] Krause, Fedar: Resection des Trigeminus innerhalb der Schädethöhle. Verhandlungen der Deutschen Gesellschaft für Chir., Berlin, June, 1892, p. 199.

way of the temporal fossa to the ganglion, doubtless their method should be considered as fraught with less danger and as offering better chances of a successful outcome than any other heretofore described, and it is noteworthy that practically by this method alone in an occasional instance has it been possible to completely extirpate the ganglion. There are many difficulties arising, chiefly from hemorrhage, which those who have seen or attempted this operation of Hartley and Krause will remember but too vividly. In the first place, the Wagner osteoplastic flap in the temporal region must be so taken that it includes the sulcus arteriosus in the anterior inferior angle—angulus sphenoidalis—of the parietal bone which lodges the middle meningeal artery, and consequently the vessel, owing to the frequent depth of the sulcus, is quite commonly lacerated and is the occasion of delay long before the real operative seat is reached. Again, when the ganglion is approached, the operator is so far—measuring on the curve of the skull—from the cavum Meckelii in which the ganglion lies, that an amount of elevation of the brain and underlying dura is necessary, which is incompatible with the preservation of the artery at its lower fixed point, namely, at the foramen spinosum. Hence, Krause finds it essential to attempt a preliminary double ligation of the vessel after its emergence from this foramen. This is a difficult procedure, and is not uncommonly unsuccessful, and hemorrhage from the meningeal under any circumstances is an unpleasant thing with which to deal.

Disadvantages other than the obligation of dealing with this arterial bugbear arise from the necessary degree of retraction which is essential for a satisfactory exposure of the deeper parts of the ganglion when this high temporal method is employed. On one occasion, in a left-sided case, a consequent aphasia was noted, and in most of the earlier operations undertaken by this method no attempt was made to remove more than the outer portion of the ganglion. Had it been deemed necessary on all occasions to expose and liberate the sensory root proximal to the ganglion, I am certain that the employment of this high temporal route would have been found frequently impossible and always attended by grave difficulties.

Even by the method which I have used, in which there is required but slight elevation of the brain, there has been invariably an asso-

ciated retardation of the pulse. This is illustrated by the accompanying ether chart, which presents characteristics almost identical with those seen on all four occasions in which I have operated.



Ether chart of ganglion case, showing "five minute" pulse-rate during operation, in illustration of compression pulse due to slight elevation of temporal lobe.

Victor Horsley[1] has proposed and carried out an operation by this route in which the dura is immediately opened and the temporal lobe itself retracted, leaving the dura covering the middle fossa in place against the bone. Few operators could handle the brain thus freed of its support without injury, and after considerable experience with the removal of the ganglion from above, even when the calvarium and brain have been removed, I have found that greater difficulties are encountered, in spite of the exposure, in freeing it from the dural envelope than will be met with when the approach is from the side, as in the operation to be described.

THE DIRECT INFRA-ARTERIAL METHOD.

The method of enucleation which the writer, with some hesitation, proposes to describe, makes use of the paramount advantage of the

[1] Horsley, Victor: The Various Surgical Procedures Devised for the Relief or Cure of Trigeminal Neuralgia. British Med. Journal, 1891, vol. ii. p. 1249.

Hartley-Krause operation, namely, that of exposure of the ganglion by the temporal route. The trephine opening through the fossa temporalis, however, is sufficiently low, so that the extradural manipulations may be conducted *underneath the arch made by the middle meningeal vessel*, which is retracted with the dura and yet remains uninjured at its two fixed points, namely, at the foramen spinosum of the temporal bone and at the sulcus arteriosus of the parietal. Under this arch with but slight elevation of the temporal lobe the entire ganglion and its sensory root may be exposed. The method may be said to give the maximum of exposure with the minimum of cerebral compression and injury of bloodvessels. The operation, therefore, differs only in its details from that proposed by Hartley and Krause; it is, however, upon detail that the success of this, supposedly one of the most delicate of surgical procedures, depends.

Anatomic Notes. There are certain anatomic features which must be taken into consideration in this particular operative method, or, indeed, in any method: in the first place, concerning the dural envelope which encloses the ganglion as well as the intracranial portion of its three peripheral branches.[1]

As commonly described, the dura splits and encloses the ganglion

[1] It is superfluous to say that an operation on the ganglion by any route should not be undertaken without perfect familiarity with its anatomic relations, and it is important that this familiarity should be gained by way of the particular method of approach selected. Only after a great number of operations at the autopsy table can one satisfactorily train his reflexes to appreciate the degree of force which it is necessary to apply at the edge of the ganglionic dural sheath in order that it may be split and the ganglion exposed by lifting away its superior covering, and similarly the force which may be applied in the subsequent elevation of the ganglion and its four branches from the underlying dura to which it has been left attached. For this procedure the only instrument which is requisite is a blunt dissector of proper shape, and in the possibilities offered by the one selected, the operator should train himself. Only after repeated operations on fresh cadavers possessing skulls of various indices did the writer feel justified in dealing with the ganglion at the operating-table and confident of removing it *in toto*. To satisfactorily free the entire ganglion is a delicate procedure and familiarity with the crackling sensations imparted to the hand while liberating it from its dural envelope cannot be overvalued. I know of no operation which could be undertaken without such preliminary experience with equal rashness. I have found the experience gained from practice on ordinary anatomic material to be unsatisfactory. The toughening of the dried dura and altered consistency of the ganglion and brain imparts, through the dissecting instrument, sensations markedly different from those which are given by fresh tissues. I have performed this particular operation on autopsy subjects about thirty times, and have removed the ganglion in a number of other ways, invariably in a much less perfect state of preservation. It is extraordinary how much easier it is to remove the ganglion through an opening in the temporal fossa by this method than it is through an open calvarium after the brain has been removed.

in a double layer as it lies in the cavum Meckelii. This fibrous envelope is closely adherent to the ganglion, and experience has shown that the point at which the layers are most easily separated is at the area of dural attachment about the superior maxillary nerve, where it enters the foramen rotundum. This is the first point at which the simple elevation of the dura is resisted, and, by careful blunt dissection, working between this point and that of its second attachment at the foramen ovale, it is found to be a comparatively easy matter to split the enclosing envelope between these two points and to elevate the upper layer of the ganglionic sheath, leaving the ganglion entirely exposed and lying attached to the basal layer of its sheath in the ganglionic fossa. There is, however, in addition to the thick dural layer of the envelope thus elevated, a second, thinner layer of transparent fibrous tissue which overlies and is attached firmly to the ganglion, extending backward over the sensory root—N. trigeminus—but which does not cover the three peripheral divisions as does the rest of the sheath. An attempt has been made to show this second layer in Fig. 2, after its separation from the ganglion, and I am ignorant as to whether this has been heretofore described. After elevation of the main dural sheath and exposure of the three peripheral branches this thin second layer is left intimately adherent to the ganglion, binding it and the sensory root to the underlying tissues. I believe this to be the chief obstacle to evulsion of the ganglion *in toto*, as it is ordinarily attempted.

The importance during the operation of leaving the ganglion attached to the underlying portion of its sheath, and of not elevating it until the final step, is due partly to the fact that it is much less difficult to free it from the underlying surface of dura and bone below than from the elastic cerebral surface, as will be emphasized later, and, furthermore, because the blood-supply of the structure comes almost entirely from beneath. The chief supply ordinarily comes from a branch of the meningeal soon after its entrance into the skull, and consequently the third division—N. mandibularis—is often the most bloody one to liberate. A small branch usually comes from the carotid and passes over the sixth nerve—N. abducens—to the under surface of the ganglion. Another common branch is a small radicle of the ophthalmic to the first division; another by way

of the foramen ovale is the lesser meningeal, and occasionally a branch appears through the foramen rotundum from the internal maxillary artery.

Relative to the middle meningeal artery, it not infrequently happens that the vessel communicates with the lachrymal artery by an anastomotic branch through the outer angle of the sinus sphenoidalis. Occasionally the latter artery may derive, in this way, its main origin from the meningeal, or *vice versa*, the meningeal from the lachrymal. Under these circumstances it can be seen that the vertical extent of the arch under which we must work is somewhat narrowed. Such an anomaly was met with in Case IV. of my series; it nevertheless did not seriously encroach the field of view.

Another factor may occasionally interfere with the view of the ganglion, one which has been encountered especially in skulls with a broad cephalic index, and this is the more embarrassing since the ganglion in such brachycephalic cases may lie from 1 to $1\frac{1}{2}$ cm. deeper than usual. This obstruction consists of a bony prominence which, in slight degree, is present in all skulls, but at times is sufficiently developed to partly hide the ganglion when viewed from the side and to ward off the curved blunt dissector in its approach to the ganglion. This process, apparently unnamed,[1] is more or less developed in all skulls and is roughly indicated by the elevation at the lower rim of the operative foramen in the drawings that accompany this paper. The process is situated to the outer side and slightly anterior to the foramen ovale on the greater wing of the sphenoid between the row of sphenoidal foramina and the sutura sphenosquamosa. In Case II., a patient with a markedly brachycephalic skull, it was necessary to chisel off the top of this prominence before the ganglion could be attacked. A similar and still more developed bony protection renders the ganglion operation in dogs almost impossible.

Underlying the lower sheath of the ganglionic envelope posteriorly there is usually to be made out a dense triangle of fibrous tissue which partly covers the foramen lacerum and under the anterior edge of which the carotid artery emerges. Over the artery and

[1] One might presume to call this the processus fasciei cerebralis ossis sphenoidalis, according to the His nomenclature.

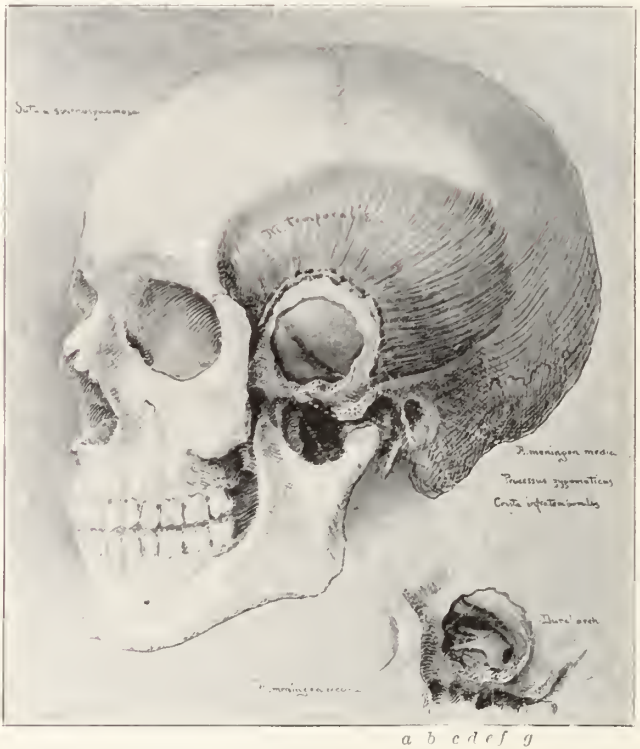
close to the edge of this ligamentous structure, and, consequently, near to the inner edge of the ganglion, the sixth nerve—N. abducens—passes. It is in this situation, on account of the diminished likelihood of injuring the sinus cavernosus, which is only expressed further forward, that I have preferred to attack the ganglion and its sensory root when it becomes necessary during the process of liberation to pry the structure outward in order to free the attachments of its inner border. I have been able in this way to avoid injury of the sinus and to remove the first division—N. ophthalmicus—by freeing it from behind forward, though I have invariably caused a paralysis—fortunately only temporary—of the sixth nerve. Injury to the sinus, however, is not such a calamity as is usually presumed. An interpretation of its character is given in *Spalteholz's Handatlas der Anatomie* (*Zweite Auflage*, 1899, Bd. ii. S. 397 und 441), which is much less startling to an operator than the manner in which it is usually depicted as resembling the other cranial sinuses.

DESCRIPTION OF THE OPERATION WITH DISCUSSION OF THE VARIOUS STEPS.

1. *Formation of muscle flap and exposure of the temporal fossa.* A horseshoe-shaped skin incision is made in the temporal region, its base about 4 cm. in breadth, corresponding to the zygomatic arch, and its convexity reaching about 5 cm. above it, but slightly higher than the level of the pinna of the ear. (See Fig. 3.) The incision, therefore, needs to be much lower, and may be considerably smaller than that for the Wagner osteoplastic procedure as adopted by Hartley and Krause. This skin flap is turned downward, some branches of the temporal artery being divided in the process, until the underlying temporal fascia is exposed well on to its attachments to the zygomatic arch and the posterior or temporal border of the malar bone. An incision is then made through the temporal fascia concentric with and just inside of the skin incision, and at the base of the skin flap it is carried along the middle of the outer surface of the zygomatic arch through the periosteum down to the bone. The periosteum is then elevated from the bony arch, leaving the masseteric attachment at its lower edge uninjured, and the zygomatic processes of the malar and temporal bones (see Fig. 1) are divided with

heavy forceps as in the resection of a rib An incision concentric with the skin incision is then carried down through the temporal muscle, and the muscle is scraped away from the bony wall of the temporal fossa, to which it has no attachment in this situation, and retracted downward together with the resected portion of the zygoma and into

FIG. 1.



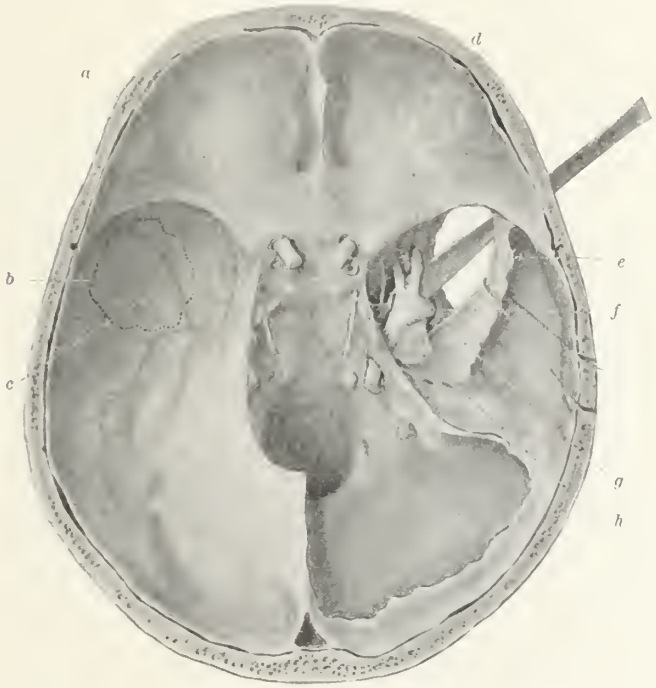
Showing relations of the middle meningeal artery to the operative foramen before and after elevation of the dura and exposure of the ganglion.

a. N. ophthalmicus. *b*. N. abducens. *c*. N. maxillaris. *d*. Underlying sheath of dural envelope. *e*. N. mandibularis. *f*. Ganglion semilunare. *g*. Foramen spinosum.

the space which this bony arch formerly occupied. (See Fig. 3.) In this way the lower portion of the temporal fossa of the skull, as far down as the attachment of the external pterygoid muscle below the infratemporal crest, is well exposed.

Discussion of Step. The one deformity consequent to the operative method via the temporal route follows upon the incision through the temporal fascia, namely, the division of the branches of the facial nerve which supply the occipitofrontalis muscle. As a result, there is a post-operative inability to elevate the eyebrow on the affected

FIG. 2.



Showing on the right, after reflection of the dura, the ganglion and its intracranial branches liberated from their dural envelope and elevated by the blunt dissector introduced through the operative foramen; on the left, the dura *in situ* and the relation of the operative foramen to the ganglion and middle meningeal artery.

a. N. abducens. *b*. Site of operative foramen. *c*. A. meningea media. *d*. Operative foramen. *e*. A. meningea media. *f*. Ganglion semilunare. *g*. Dura mater (reflected). *h*. Dura propria of ganglion.

side. This paralysis, however, is not apparent during expressional rest except in old individuals in whom there may be some resultant smoothing out of the cutaneous wrinkles on that side. This deformity is well shown in the photograph of one of Krause's patients.

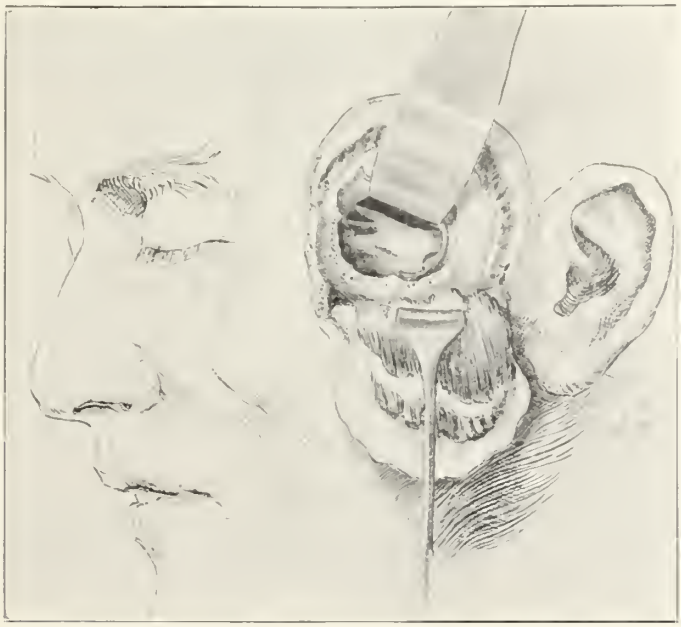
(Op. cit., S. 44, Abbildung 14.) I have found that it is necessary to temporarily reseet the zygoma in order to satisfaetorily retract the temporal muscle. A downward displacement of the bone, of only a centimetre or two, is all that is required to make room for the muscle, whieh otherwise would areh up over the zygoma and prevent proper exposure of the lowest portion of the fossa temporalis. It is easier to preserve than to detach the bony fragment, on aceount of the firm attaehment of the masseter. This is not a neeessary detail, however, and perhaps it would be advantageous to remove it. A new zygoma in one of my eases has re-formed after excision, and possibly in this case there is post-operative deformity, sinee the atrophy of the temporal and masseter muscles shows less plainly than in the other eases in whieh the arch of the replaced zygoma seems unduly prominent on account of the sunken fossæ above and below it.

2. *Exposure and elevation of dura as far as the ganglionic sheath.* With a mallet and gouge a small trephine opening is made through the most prominent portion of the exposed great wing of the sphenoid, and with rongeur forceps this opening is enlarged to a diameter of about 3 cm., its lower margin being earried well down and possibly including the ridge between the temporal and zygomatic fossa —the erista infratemporalis. The uninjured middle meningeal artery runs on the dura thus exposed, across the opening in the bone as the diameter of its cirele. (See Fig. 1.) The dura, with this vessel, may then be easily lifted, almost bloodlessly, from the base of the middle fossa until the first point of firm dural attaehment at the foramen ovale is reaehed.

Discussion of Step. It has seemed unnecessary to the writer to attempt to make an osteoplastie flap even were it possible to deal with it in this deep situation, for the reason that the opening to be made through the skull is so small and after elosure of the wound so well proteeted. It is possible, indeed, that new bone is subsequently formed to eover the defeet, sinee the slight pulsation of the flap which is apparent for some time after the operation ultimately disappears. It is well to make the first opening in the bone at the upper part of the proposed area of removal, sinee it is easier to bite away the bone with the rongeur foreeps in a downward direction,

and, furthermore, because as the base is approached the sphenoidal wing becomes much thicker—occasionally 5-6 mm.—and, consequently, is less easily penetrated with the mallet and chisel. Care must be taken in the use of the gouge, since the bone is at times exceedingly thin and one or two strokes with the mallet will carry the instrument through. Additional care is necessary, for the middle meningeal artery is usually exposed by the first small opening in

FIG. 3.



Sketch from cadaver of the field of operation: showing situation of the incision, retraction of temporal muscle over displaced zygomatic arch, osseous foramen and final exposure of ganglion under meningeal arch.

the skull. It ordinarily runs beneath the most prominent part of the wing of the sphenoid and squamous portion of the temporal bones, which have been uncovered, and this is naturally the point selected for the primary opening.

It is important, in view of preservation of the ganglion and avoidance of possible injury to the deeper vessels, that no fragments of bone

be allowed to fall down between the skull and dura, since at a later stage of the operation, when it is necessary to firmly press pledgets of gauze against the ganglion to check the bleeding from its underlying arterioles, these spiculæ may, as occurred in one of my cases, be firmly driven into its substance. By a similar accident one of them might be driven into the sinus. Occasionally the prominence, which has been described above, as present on the floor of the fossæ, may be of sufficient size to later interfere with a satisfactory approach to the foramen ovale. This may then be chiselled off or the floor of the fossa rongeured away so as to include it. In the drawings I have not made the trephine opening quite low enough. It should be carried down so as to include the crista infratemporalis, which, therefore, should not be shown preserved as in Fig. 1.

3. *Elevation of dura with meningeal artery and exposure of upper surface of ganglion.* By careful blunt dissection with the proper instrument, and by working at the dural attachment about the foramen rotundum and in the line between this point and the foramen ovale, where it is again firmly attached, the edge of the dural envelope which encloses the ganglion and its peripheral intracranial branches may be split, and by careful elevation the entire upper surface of the stellate structure be exposed well back on to the sensory root, the ganglion being left in its bed still adherent to the underlying portion of the envelope.

Discussion of Step. This procedure should be attended with but little hemorrhage, since the blood-supply, as heretofore stated, is from below. It is of importance that *the ganglion should not be elevated in this manœuvre*, since it is advisable to postpone what degree of hemorrhage is unavoidable as long as possible, and, furthermore, because it is much easier to elevate the overlying dural sheath from the ganglion if its attachments to the unyielding base have remained uninjured. This entire procedure is carried on under the arch made by elevating the temporal lobe and its overlying dura and artery. A simple spatula of about $2\frac{1}{4}$ cm. in width, which can be bent at the proper angle, makes the most satisfactory retractor for these structures. (See Fig. 3.)

4. *Liberation and extraction of ganglion and its branches.* After the exposure of the upper surface of the ganglion and before

division of any of the peripheral branches, these three nerves with the ganglion and trigeminal root should be liberated from the attachments to the base. (See Fig. 2, right side.) This is readily accomplished by working with the blunt dissector in the crotches between the second and third divisions and also along each side of the nerves. After the ganglion and the second and third divisions have been liberated and can be lifted up by the dissector, it is necessary to free the superior and internal edge of the trigeminal root and first division. It is well to conduct these manipulations as near as possible to the sensory root, since that is the safest point and one at which there is less likelihood of injuring the cavernous sinus and sixth nerve. The ganglionic structure may thus be completely liberated (as shown in Fig. 2) without division or laceration of a single branch. With a firmly locking pair of hæmostatic forceps—I have used Kocher's—the structure may then be grasped just back of the site of the true ganglion on the trigeminal root; the three peripheral divisions are in turn held up with a blunt nerve hook and divided with scissors close to their foramina; the sensory root is then evulsed by means of the previously attached pair of forceps.

Discussion of Step. This part of the operation is the most difficult and the one in which preliminary training on the cadaver is found most essential. The degree of force necessary to separate the ganglion without injuring it, for if lacerated or torn away from its roots its extraction becomes most uncertain, can only be learned by experience which should hardly be gained at the expense of the patient. The operator's reflexes should have become familiarized with the crackling sensations imparted to the hand on separating the adhesions at one point or another during the process of liberation. The bleeding which follows on the manœuvre, especially about the foramen ovale, is sometimes very annoying, but ordinarily may be checked in a few minutes by the pressure of a pledget of gauze. It would naturally be supposed that the proximity of the meningeal and its fixed point at the foramen spinosum would be an embarrassment, especially when an attempt is made to liberate the third division by insinuating the elevator under the posterior border of the nerve—N. mandibularis—between it and the artery. This I have not found to be the case.

I do not see how the third and fourth nerves can be injured in this procedure; the sixth, however, lies very near the ganglion, has always been seen, and, I must confess, injured in each of my four cases. This, however, has fortunately occasioned only temporary symptoms, the resulting internal strabismus having disappeared in each case in the course of a few weeks. In one instance I felt certain that I tore the nerve across; if so it must have regenerated. There has also resulted in all of my cases a temporary paralysis of the sympathetic with contracted pupil. This has invariably disappeared much earlier than the motor paralysis of the abducens. As stated above, it is well in the liberation of the first division—N. ophthalmicus—to free the nerve at the ganglionic end and to strip it out from behind forward if it is desired to remove it at all, else the cavernous sinus may be injured. This accident, however, is by no means such a calamity as it is credited with being. The sinus is not an open canal, as usually believed, but made up of compartments in which local thrombosis may occur readily and promptly, and thus hemorrhage be controlled by a few moments of pressure. Bleeding from the small ganglionic arteries about the foramen ovale may be much more annoying. On one occasion I accidentally plunged the dissector directly into the sinus anterior to the ophthalmic border of the true ganglion. The profuse hemorrhage ceased after a few moments of pressure with some gauze. The trigeminal sensory if properly extracted invariably comes away from the pons, where it is loosely attached. I have never seen any evidence of shock consequent upon this procedure such as Horsley describes in his single case.

5. *Closure and Dressing.* The zygoma and flap of skin, muscle and fascia are replaced. The zygomatic arch in my first case, as stated, was removed, and in one of the later ones wired in position. This is an unnecessary detail, inasmuch as the masseter is paralyzed and the resected portion of the bone when replaced remains in position. The temporal muscle and fascia are secured in place by fine, interrupted sutures at the upper curve of the incision, and a few sutures are taken in the divided periosteum and fascia over the replaced zygomatic arch. In applying the dressing the eye is covered by a large sheet of rubber protective which bridges across from the

nose and forehead to the malar prominence of the cheek and prevents the pressure of the bandage against the eye. In none of my four cases has the wound been drained, and in none has there been failure to obtain healing by primary union; the resulting scar has

FIG. 4.

Photograph of Case IV., taken on the tenth day, in illustration of a method of protecting the eye.

been very slight (see Fig. 4), and in one instance hardly to be detected. An unsightly scar, such as is shown in many photographs of cases, would almost deter a surgeon from the operation.

I have not found it necessary to suture the lids as has been advo-

cated in treatment of the eye. In fact, I should think the local reaction resultant to this procedure would be detrimental in case there was an ensuing keratitis, which apparently is at times unavoidable. The simple protective method which has been used is shown in the accompanying photograph of one of the patients. The eye is thus kept moist, protected from dust, and the patient may dimly see through the covering. A Buller shield answers the same purpose.

It is not the writer's intention on this occasion to take up the physiologic aspects of the four cases which have been operated on, nor to report the histories in any detail, except in so much as they may be of value in interpreting the pathologic lesions, to be described by Drs. Barker and Spiller. The physiologic question relative to disturbances of taste and of secretory activity in the trigeminal area, the consideration of the post-operative areas of sensory anæsthesia, the discussion of the so-called trophic changes consequent on removal of the ganglion, and similar topics, must be left to a subsequent communication, in spite of the fact that they doubtless represent the most interesting side of the Gasserian ganglion question, and one which is second only in importance to its pathologic aspect. The photographs show that in each instance the ganglion and intracranial roots have been completely removed and the resulting anæsthesia has been absolute over the entire area of trigeminal sensory distribution in consequence. The completeness of the post-operative anæsthesia may be regarded as an index of the totality of the extirpation.

It will be noted that the ganglia of the first two cases examined by Dr. Spiller were removed during a period of extreme exacerbation of the neuralgia. In Dr. Barker's cases the operation was performed during a period of comparative freedom from pain. Brief summaries of the histories preceding the operative period are as follows:

Case I.—James W.. aged sixty-three years, entered the hospital August 2, 1899. He had been a sea captain by profession until the onset of his right-sided neuralgia, from which he had suffered for ten years. The pain occurred originally in the third division of the trigeminus. In July, 1896, and again in June, 1897, two peripheral operations with evulsion of the infraorbital and inferior dental

nerves had failed to give him more than a few weeks of respite from pain. For the two years before entrance he had been bed-ridden, and his sufferings had had no remission. At the time of operation his extreme paroxysms occurred every minute and a half on an average, and with only slight relief during the intervals. His chief point of radiation of pain was just below the outer corner of the mouth. From there the pain spread into the territory of all three divisions of the N. trigeminus during the paroxysms, to terminate at a point near the parietal eminence. The ganglion is shown in the accompanying photograph (Fig. 5).

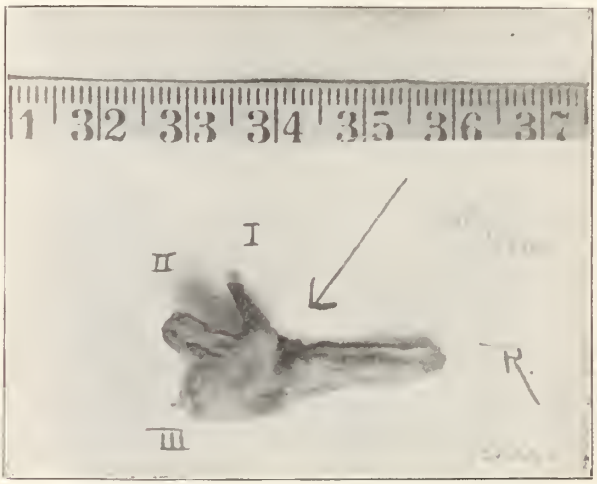
FIG. 5.

Specimen from Case I. (Dr. Spiller.) Photograph was taken from the fresh tissue, and consequently shows points of high light. The first division (I.) (N. ophthalmicus) was removed as a separate piece after extraction of the ganglion and other branches. The tissue was placed in Müller's fluid one-half hour after removal.

CASE II.—W. E., aged fifty-five years, entered the hospital December 17, 1899. He had been a business man until neuralgia interrupted his activities. He had suffered from pain, which appeared primarily in the distribution of the infraorbital branch, for

twelve years. In 1892 the infraorbital nerve was evulsed, with relief for several months. On the return of pain, the inferior dental became involved, and subsequently the supraorbital region, and pain finally extended to the territory innervated by the suboccipital nerves. His sufferings for several months before entrance were such that he had been in confinement, and, on admission to the hospital, he was practically maniacal. His chief pain-point from which the paroxysms radiated was situated in the right nasolabial fold. His

Fig. 6.



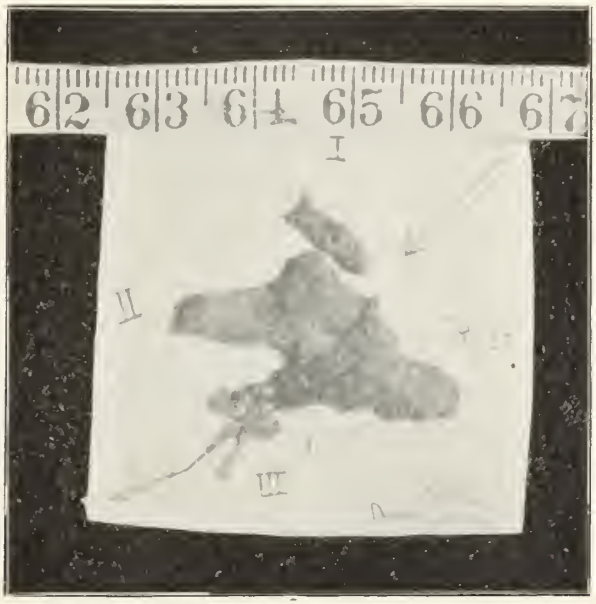
Tissues removed from Case II. (Dr. Spiller.) The arrow indicates the direction of application of the evulsing forceps, whose imprint shows upon the ganglion. Photograph taken from specimen after hardening in formalin, and high lights as in Fig. 5, from reflection of fresh tissues, do not appear.

sufferings were continuous, with exacerbations every few moments, or consequent on the slightest peripheral stimulus. The ganglion and branches are shown in the accompanying photograph (Fig. 6).

Case III.—A. D., aged thirty-eight years, a shoemaker by trade, entered the hospital in January, 1900, having suffered from trifacial neuralgia for only two years. The onset was attributed to exposure and pain, and was at first limited to the supraorbital area. One year later the infraorbital region became involved and soon the whole

territory of the third division, the pain during the paroxysms extending into the suboccipital region. In March, 1899, by peripheral operations, I evulsed an inch or two of the inferior dental and infraorbital nerves, which showed the usual histologic changes after being removed. After relief for eight months the pain returned—November, 1899—with renewed vigor. The paroxysms at the time of

FIG. 7.

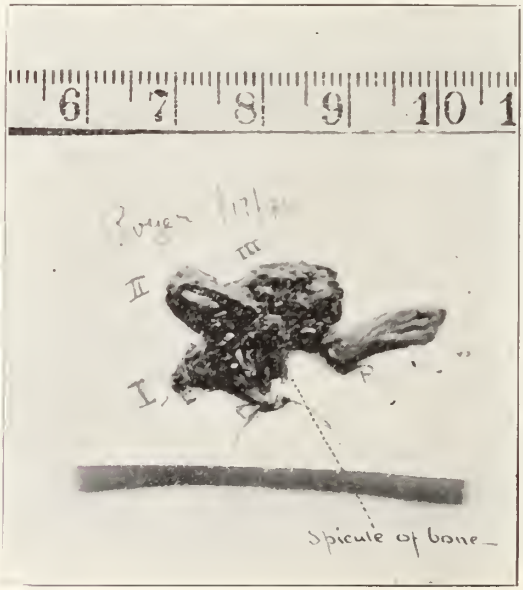


Specimen from Case III. (Dr. Spiller.) The arrow points to the line of impress of the evulsing forceps. The third divisiou (N. mandihularis) is considerably lacerated, as an unsuccessful attempt was made to evulse this from the foramen ovale instead of dividing the root as usual. Shreds of the motor root can be seen distinct from the rest. The first division (N. ophthalmicus) was torn away from the ganglion during its elevation and was removed from the wall of the sinus after extraction of the ganglion. Photograph taken after hardening in formalin.

entrance were not frequent nor very severe. The pain spread downward from the supraorbital division into the territory supplied by the second and third branches. The patient was operated on during this interval of comparative freedom. The ganglion is shown in Fig. 7.

Case IV.—Elizabeth R., aged sixty years, had suffered for seven years from left-sided tic douloureux, which originated in the superior maxillary branch of the trigeminal nerve, and was attributed to exposure. The definite point of origin of her paroxysms had always been situated near the ala of the nose. In August, 1897, 5 cm. of the infraorbital nerve were evulsed (Cushing) from the floor of the orbit. The nerve histologically showed the usual degenerative changes. Relief ensued for sixteen months, after which interval,

Fig. 8.



Specimen from Case IV. (Dr. Barker.) A spicule of bone is shown driven firmly into the ganglion, which was not removed in a good state of preservation. The sensory root (R) (N. trigeminus) tore away from the ganglion during extraction and was subsequently removed. Photograph taken after hardening in formalin.

with the return of pain, the first and third divisions became invaded. In June, 1899, the regenerated nerve from the same situation was again evulsed (Mitchell), with subsequent relief for only four months. In January, 1900, the ganglion and roots were removed *in toto*, though with considerable difficulty and not in a good state of preservation. (See Fig. 8.) The operation was performed during a period of comparative freedom from pain.

METHOD OF EXPOSING THE GASSERIAN GANGLION: REMOVAL OF THE SUPERIOR MAXILLARY NERVE.

By GWILYM G. DAVIS, M.D., M.R.C.S. Eng.,
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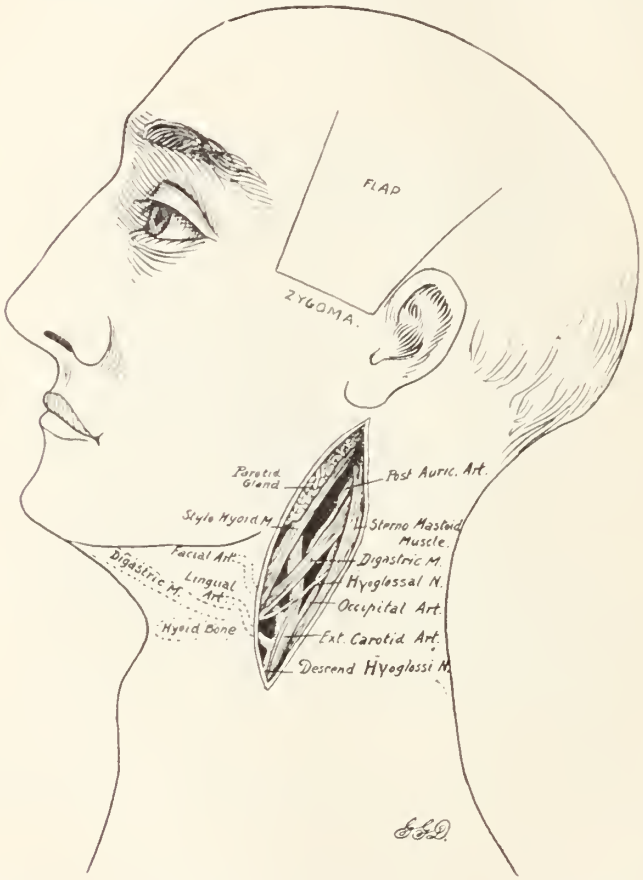
[Read at a special meeting held April 20, 1900.]

This patient, about sixty years of age, is brought before you to illustrate certain methods of procedure in operating on the Gasserian ganglion. He was affected with neuralgia confined almost entirely to the superior maxillary nerve. For this condition several operations had been done on the face, but the pain having returned I performed Carnochan's operation on Meckel's ganglion. An opening was made in the anterior wall of the maxillary sinus, the posterior wall was chiselled through and by means of hooks and curettes the ganglion was broken up and removed as much as possible. He remained free from pain for three or four months and then had a return. The question arose as to whether an attack should be made on the Gasserian ganglion itself or on the superior maxillary nerve within the skull. The removal of the ophthalmic branch with the ganglion is now, I believe, avoided when possible, and as both it and the inferior maxillary divisions were free from pain I decided to limit my interference to the superior maxillary nerve.

The main difficulties experienced in the operation have arisen from the hemorrhage encountered, and it was to this question that my attention was mainly directed. The bleeding in cutting the flap is considerable, as is also that from the middle and small meningeal arteries and the veins, including the cavernous sinus, in the region of the ganglion. Any bleeding from the sinus was obviated by not

interfering with the ophthalmic nerve. To control the arterial bleeding it was decided to ligate the external carotid artery. This procedure was, I believe, suggested by Dr. Fowler.

In ligating the external carotid artery the ligature was placed around it just above the digastric muscle. (See illustration.) The



incision was placed behind the angle of the jaw along the anterior edge of the sternomastoid muscle. This was pulled backward and the parotid gland pushed forward. The veins contributing to form the external jugular were pulled forward and the oblique fibres of the digastric muscle came into view; a little parting of the tissues

then enabled one to see the hypoglossal nerve below the muscle and winding around the occipital artery to its outer side; to the inner side, coming from the trunk of the external carotid, were the commencement of the facial and lingual arteries. The ascending pharyngeal artery is posterior and may not be seen. Just above the digastric muscle is seen the stylohyoid muscle, and the posterior auricular artery, comparatively small, may be seen winding along the upper edge of the digastric muscle. The stylohyoid muscle was pulled upward, the digastric downward, and the ligature of catgut placed between them on the external carotid artery. This allowed the blood still to ascend to the scalp through the occipital and posterior auricular arteries.

To cut the usual Hartley-Krause horseshoe-shaped flap with its base on the zygoma would be to court sloughing of the flap if the main blood-supply, through the temporal artery, was cut off by ligation of the external carotid artery. Necrosis of the bone and even sloughing of the flap itself have been known to occur. To avoid this a flap of the shape of a truncated cone was cut with its base upward and small end downward. Two longitudinal cuts were made upward, one from the junction of the anterior edge of the ear and the zygoma and the other from the junction of the anterior and middle thirds of the zygoma; these were joined by a transverse cut along the zygoma. This flap embraced the skin and superficial fascia down to the temporal aponeurosis.

The temporal muscle and aponeurosis were then cut in the lines of the two lateral incisions, but the transverse cut was made high up near the temporal ridge. This muscle, having been thus loosened above, was pushed down beneath the zygoma. The skull was opened by a trephine and the opening enlarged by the rongeur forceps. In biting away the bone the middle meningeal artery was torn, but gave rise to no troublesome bleeding. The dura mater and brain were then lifted and the superior maxillary nerve exposed as it ran from the Gasserian ganglion to the foramen rotundum. Grasping the nerve, as it entered the bone, with a hæmostatic forceps, it was pulled upward. With another forceps it was grasped closer to the ganglion and twisted loose from it. The bone was not replaced, but the temporal fascia and muscle were drawn upward and sutured in

place by catgut. The flap was then brought down and sewed in position.

The bleeding during the operation was at no time troublesome. As to the subsequent course, the wounds healed by primary union; there were no circulatory disturbances. The opening in the skull is closed by firm, fibrous tissue and the pain has disappeared. The non-replacement of the bone was suggested by Professor Tiffany, and this case demonstrates that the brain is sufficiently protected without it.

ENDOTHELIOMA OF THE GASSERIAN GANGLION. TWO SUCCESSIVE RESECTIONS OF THE GANGLION: FIRST, BY THE EXTRADURAL (HARTLEY-KRAUSE) OPERATION: AND, SECONDLY, BY AN INTRADURAL OPERATION.

CLINICAL REPORT BY F. X. DERCUM, A.M., M.D.,
PROFESSOR OF NERVOUS AND MENTAL DISEASES, JEFFERSON MEDICAL COLLEGE, NEUROLOGIST TO THE PHILADELPHIA HOSPITAL.

SURGICAL REPORT BY W. W. KEEN, M.D., LL.D.,
PROFESSOR OF THE PRINCIPLES OF SURGERY AND OF CLINICAL SURGERY, JEFFERSON MEDICAL COLLEGE, PHILADELPHIA.

PATHOLOGIC REPORT BY WM. G. SPILLER, M.D.,
PROFESSOR OF DISEASES OF THE NERVOUS SYSTEM IN THE PHILADELPHIA POLYCLINIC, PHILADELPHIA.

[Read at a special meeting held April 20, 1900.]

Dr. Dercum's Clinical Report.

The following case, both because of the rarity of the affection from which the patient suffered and because of the interesting nature of the symptoms, is worthy of record:

Mr. X., a business man, thirty-two years of age, married, a native of Louisville, Ky., came under my care on November 20, 1899.

Family History. The father died at sixty-eight years of age, of some affection of the heart. The mother is living and well at sixty, also a brother and sister. The family history is negative as regards mental, nervous and other diseases.

Personal History. The patient was rather delicate as a baby, suffered from various diseases of childhood, had an attack of scarlet fever which resulted in disease of the ears, but in youth he seemed

to be rather healthy. Twelve years ago he was said to have had an attack of typhoid fever. Some ten years ago he suffered from a venereal sore which was believed to be syphilitic, and although he never presented any secondary symptoms, he was for some time at Hot Springs, Ark., under treatment. The physician at that place, so we were informed, thought he did not have specific disease. He had also acquired gonorrhœa on two occasions. When twenty or twenty-one years of age he went into business as a manufacturer, and worked very hard, often day and night. He was a man of extremely nervous temperament, and was married in September, 1895. Subsequent to this time he complained greatly of severe shooting pains in his legs, and he occasionally walked with a limp. The pains were regarded as rheumatic, and he suffered from them in two successive winters. He had inflammation of the bowels in 1898 and was ill at this time some six or eight weeks. Subsequently he appeared to be well, very strong and energetic, but for over a year past has complained of being tired at night and always wished to retire early. He would sleep heavily and without awakening during the entire night. General weakness became evident about this time and was very marked. At times he would have very severe headaches, especially in the mornings, but they would pass off after he had been up and around for an hour or two.

In February, 1898, he discovered a glandular swelling on the left side of the neck, about the size of an olive. It remained stationary for about eighteen months. Early in the summer of 1899 he began to lose in weight and to be extremely irritable and nervous. Various physicians who were consulted considered the glandular swelling tubercular, except one who thought it syphilitic and advised treatment at Hot Springs. He went there on July 1st. About the time of his arrival he began to suffer from very severe pain over the left temple and over the left side of the top of the head. The swelling on the side of the neck also became painful; it had not pained him previously. It now began to grow, and grew rapidly until it attained the size of a large egg. The pain in the temple was much worse in the evening and night, the patient not being able to sleep until about four or five o'clock in the morning. He took the baths and also had mixed treatment for seven weeks, while the swelling

was painted with iodine. No relief whatever was obtained from this treatment. He then consulted Dr. Abbe, of New York, who removed a series of glands, varying in size from an olive down. These glands, as was subsequently learned from Dr. Abbe, were of a gray, even consistence, not unlike tubercle, but entirely without caseation. A microscopic examination was made at the College of Physicians and Surgeons of New York and endothelioma diagnosticated. Dr. Abbe very kindly sent me a section of one of these glands, and I had no difficulty in confirming the diagnosis. The pain in the side of the head subsequently assumed the character of trifacial neuralgia and grew steadily worse. He was treated ineffectually with large doses of the iodides, and morphine finally had to be resorted to.

On November 1st, Dr. Abbe operated on him a second time, now removing the infraorbital division of the fifth nerve. The pain continued, though it appears to have been for a time less marked in the distribution of this branch. His suffering was so great that he was transferred to a sanatorium. Here his treatment was also without relief.

During the period of development of the neuralgia he had some recurrence of the shooting pains in the legs. For two months he had been in the habit, according to his wife, of dropping off to sleep suddenly—for instance, while talking he would suddenly fall asleep. This condition was for a time quite marked. The wife herself attributed it to the great loss of sleep at night, thinking that he simply fell asleep in this way during the day from sheer exhaustion.

When first seen by me, November 20, 1899, he was evidently suffering greatly from pain in the entire trigeminal distribution of the left side. The suffering was so terrible that he could not at this time be carefully examined. It was only after a certain amount of morphine had been administered on the following day that an examination could be made. Even after one-quarter grain of morphine the pain was agonizing in character and constant, there being, according to the statement of the patient, not a single instant of relief. His expression, gestures and conduct evinced a degree of pain and suffering greater than I have ever before witnessed. The pain appeared to be most marked in the temple, the eyeball and the tissues about the orbit and the brow, less marked in the infraorbital distribution

and least in the inferior maxillary distribution. The patient constantly kept the left eye covered by his left hand, and maintained a loud and almost ceaseless moaning and groaning. The suffering, as just stated, appeared to be most marked in the orbit and brow, though at times the patient said the greatest pain was present in the teeth of the upper jaw, more especially in the incisors. He often begged piteously for something to be done to the teeth to relieve them. The pain was accompanied by irregular recurring twitching

FIG. 1.

Area of hypæsthesia previous to first operation by Professor Keen. The dark patch represents the area in which hypæsthesia was pronounced.

of the zygomatic muscles. Occasionally the twitching also involved the elevators of the upper lip and of the angle of the mouth. Tested for sensation it was found that the entire area of the trigeminal distribution to the face was decidedly hypæsthetic. This hypæsthesia could readily be demonstrated over the left side of the nose, the left brow and forehead and over the left temple and cheek. It was especially pronounced over an oval patch in the infraorbital distri-

bution. This patch, which appeared to have the infraorbital foramen as its centre, was about one and a half inches in its vertical diameter and one inch in its transverse. (See Fig. 1.) Sensation was everywhere sufficiently preserved for the patient to distinguish between a pin point and the head of a pin. Pressure sensation appeared to be everywhere well preserved. This was also the case with the temperature sense. The superficial pain sense could not be studied accurately, as the patient declared all handling of the parts gave him pain. Distinct pricking of the skin, however, gave more decided demonstrations of pain than a mere touch, and there can be no doubt that marked hypæralgesia was present. The conjunctiva was distinctly hypæsthetic as compared with the conjunctiva of the opposite eye. The condition of the sense of taste on the left side of the tongue could not be satisfactorily determined. Movements of the tongue and the opening of the mouth so greatly increased the suffering of the patient that no test could be applied with satisfaction. The pain, it should be added, was referred by the patient not only to the surface but also deeply into the tissues.

The patient was excessively weak, and, according to his own statements, had lost greatly in weight. He stood with some difficulty, and when prevailed on to walk did so with a slightly staggering gait. When tested for station with feet apposed and with his eyes closed, he evinced a tendency to fall backward. These phenomena impressed me at the time as being rather the symptoms of extreme weakness than those of ataxia. When he was seated, the movements of the arms were tested and no ataxia of these members was revealed. The knee-jerks, however, were both absolutely lost. They could neither be elicited by the ordinary methods nor by efforts at reenforcement. The general examination failed to reveal any sensory losses whatever, nor was there any sensory retardation. It should be added that the patient also suffered from an old cystitis which occasionally gave rise to considerable pain.

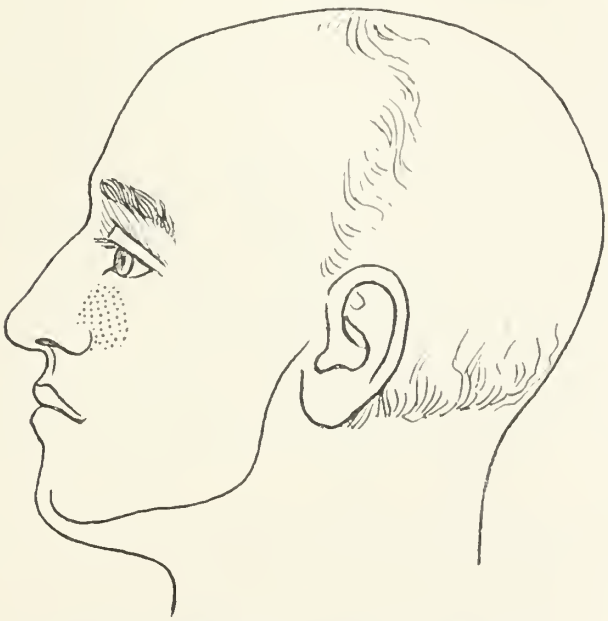
Professor G. E. de Schweinitz made an examination of both eyes, with the patient in bed, his head and shoulders raised on pillows. The pupillary reflexes were normal. There was no failure in rotation of the eyes and no nystagmus. Each disk was somewhat anæmic. There was neither congestion, neuritis nor atrophy. Both

retinæ were normal, the retinal circulation also, and fields for white and red. In addition there was present chronic otitis media of the right ear. This was later confirmed by a detailed examination of the ears, made by Dr. Walter J. Freeman, who reported in addition that the membrane of the left ear was decidedly depressed and scarred, but could readily be inflated.

The non-paroxysmal character of the neuralgia, its frightful severity and unvarying intensity, the deep-seated temporal pain, the nearly simultaneous involvement of all branches of the trifacial, the hypæsthesia in the distribution of the fifth nerve, and the somnolency suggested to me both an organic cause and the Gasserian ganglion as the seat of the disease. Further, the remarkable result of the microscopic examination of the glandular masses, removed from the neck by Dr. Abbe, suggested the existence of a neoplasm within the cranial cavity. It would be extremely difficult to explain the presence of an endothelioma in the lymphatic glands of the neck on other grounds; and it, therefore, seemed to me a most important point to confirm the diagnosis as to the nature of the tumors removed by Dr. Abbe by a personal examination, and this, thanks to the kindness of the Doctor, I was able to do. On November 23d I called in Professor W. W. Keen, with a view to a possible surgical operation directed to the Gasserian ganglion. Dr. Keen carefully examined the patient and was greatly impressed by the character of the pain, which differed, as he expressed it, from that in any case of trigeminal neuralgia he had before seen. On November 25th he was removed to Dr. Keen's private hospital, and on November 27th an operation was performed in which, as the Doctor details, he discovered and removed in large part a tumor occupying the position of the Gasserian ganglion. As far as could be determined, at the time, the foramen rotundum had become both divided and obliterated. Much of the mass back of this and also over the foramen ovale and to some extent posterior to the latter was removed. The fragments of the tumor resembled fibrosarcoma in their microscopic appearance. They were handed to Professor W. G. Spiller for microscopic examination. It was thought wise to discontinue the operation at this point. The patient promptly rallied, but on resuming consciousness at once complained as bitterly as before of

pain. It was also evident that he was delirious, and delirium accompanied by great excitement continued for about ten days, during which it was impossible to make an accurate examination as to the sensory phenomena. He was excessively noisy and loud and almost incessant in his lamentations. Some quiet followed hypodermatic injections of morphine, but the periods of relief thus secured were generally short and imperfect.

FIG. 2.



Area of hypæsthesia after first operation by Professor Keen.

He made a good surgical recovery from the operation, and the delirium somewhat abated, but the pain continued with undiminished severity. He was examined on December 13th, with the very surprising result that no change whatever in the sensory phenomena was observed, except some increase in the hypæsthesia. The temple and the cheek were now decidedly more hypæsthetic than before. (See Fig. 2.) This increased diminution in sensation could not, however, be claimed for the conjunctiva or for the brow. The patient

was still everywhere able to appreciate touch, and he could for the most part distinguish a pin point from a pin head, though occasionally he failed to make the distinction—especially was this true over the oval patch of pronounced hypæsthesia in the infraorbital distribution discovered at the first examination. He could still everywhere readily distinguish between a hot and a cold spoon, and also between light and deep pressure. An examination of the sense of taste was again without definite result; the patient still complaining bitterly of the pain in his upper teeth and of great distress when any manipulation involving the mouth was attempted. He did say, however, that his food did not taste properly and that the left side of his throat was sore. The left masseter and temporal muscles were completely paralyzed. Some time after the operation a paresis of the left rectus externus was also observed. Professor G. E. de Schweinitz again examined the eyes and reported a complete paralysis of the left external rectus muscle, but no other changes.

The pain had evidently not been appreciably influenced by the operation. Professor Spiller further reported that he had found only a few ganglion cells in the fragments of the tumor which had been handed him, and after repeated consultations with Dr. Keen and also with Dr. Charles K. Mills, it was decided to again submit the patient to operation. The second operation was performed by Dr. Keen on December 26th. The details and extent of this are described by Dr. Keen. Suffice it for me to say that the operation, as far as it was possible to judge, was most thorough and extensive. All accessible portions of the tumor, except that in relation with the cavernous sinus, were removed. In addition part of the inner end of the petrous bone, including the depression normally occupied by the Gasserian ganglion, was chiselled away and the anterior surface of the bone freely exposed.

As before, the patient rallied well from the operation, but he again passed into a condition of confusion and delirium. He was, as before, noisy, and loudly and ceaselessly dwelt on his pain, which seemed to be unabated. As before, it was referred to the orbit and brow and to the upper teeth. It was not until four days had elapsed that it was possible to make another sensory examination. It was then found that the increased hypæsthesia noted at the first examination

now involved the entire trigeminal distribution. It was, however, as before, merely a hypæsthesia and not an anæsthesia. The patient was still able to appreciate contact, still able to appreciate decided differences in pressure, and, what I regarded as extremely remarkable, still able to appreciate differences in temperature. He readily distinguished, and always correctly, between a spoon dipped in hot and another dipped in cold water. The hypæsthesia was as before most pronounced in the oval patch in the infraorbital distribution noted at the previous examinations. In this area the patient would at times fail to differentiate between simple contact and pin pricks, but total sensory loss did not exist. The hypæsthesia of the left side of the face further was not sharply limited by the middle line, but merely seemed to grow less pronounced as the sound side was approached. For instance, he felt a pin point clearly on the right side of the nose and for some distance after the bridge of the nose had been well crossed over to the left side. This fact was also observed in the lips, the chin and the forehead. At the final examination the conjunctiva appeared to be profoundly hypæsthetic but not anæsthetic. It was difficult, however, because of the patient's lamentations and excitability, to conduct this portion of the examination satisfactorily. For the same reason and because of the intense pain which the patient was suffering, no attempt was made to study the sense of taste at this time. The above observations, as before, were verified repeatedly.

Subsequently little or no change took place in the patient's condition, and his wound having healed by January 9th, he left the city for his home, still complaining loudly and constantly of his pain, and still holding his hand over his brow. I have been informed that since his return home the pain has continued, and that he still requires morphine for its relief. The cornea is now absolutely anæsthetic. The eye is immovable and blind and there is much difficulty in raising the upper lid. His general health is much improved, though he is quite anæmic and very thin. Recently another enlarged gland has appeared under the jaw on the left side.

Remarks. This remarkable case is interesting, first, because of the persistence of sensation after undoubted extirpation of the Gasserian ganglion; and, secondly, because of the rarity of tumors in-

volving this structure. The persistence of sensation would perhaps at first sight suggest that the ganglion and its branches had been imperfectly removed, but no one present at the time of the operation could doubt the accuracy of the anatomic verification nor the thoroughness with which the work was done.

Persistence of sensation, however, both after removal of the branches and even after extirpation of the ganglion is not unknown. Dr. John K. Mitchell[1] has reported two most interesting cases, in the first of which portions of the supraorbital and supratrochlear nerves were removed by Dr. Keen. Subsequently some degree of anæsthesia was present for touch and pain, but it was noticed that the loss of sensation was less complete than might have been expected, and seemed to be absolute only in an oval area at the outer canthus of the eyelids and of the upper lid. In the second patient Dr. Keen resected the supraorbital and the infraorbital nerves. A subsequent examination revealed an even less degree of loss of touch, pain and heat sense than was present in the first case. "Indeed," says Dr. Mitchell, "it could hardly be said that there was more than slight delay or impairment of perception anywhere in the supraorbital, nasal, palpebral or labial branches." When the patient was discharged after the ninth day there was neither slowness nor impairment of sensation for any form of stimulation. "The touch of the finest filament of thread was instantly felt and correctly located everywhere on the cheek, temple, nose, eyelids and upper lip." Dr. Mitchell also reports his findings in a case in which Dr. Keen had removed the Gasserian ganglion: "After the removal of the ganglion the patient's mental condition was such for nine or ten weeks that no study of the sensation could be made, but when it became possible there was no absolute anæsthesia to be found except between the margins of the wound. The touch sense was everywhere preserved in some degree, the pain sense was but slightly less in degree than before the operation." "The mucous membrane of the lips and cheek of the right side and of the right side of the tongue was also partially anæsthetic." On the right side the sense of taste was entirely lost. In explanation of these remarkable findings, Dr.

[1] Mitchell, John K.: Journal of Nervous and Mental Disease, June, 1898.

Mitchell suggests the presence of sensory fibres in the facial nerve, and cites the statements of Turner and Ramon y Cajal as to the relations of the facial nucleus to the sensory trigeminal root and to the substantia gelatinosa. He also cites Frankel-Hochwart's results as to the evidence of slight sensory losses present in some cases of paralysis of the facial nerve. The thought also suggests itself to me that in a case like the present, in which the Gasserian ganglion is slowly and gradually involved, that perhaps a *gradual* substitution of function may take place through the facial nerve.

The persistence of the pain subsequent to the operation is perhaps to be referred to degenerative changes in the sensory root of the fifth nerve, and perhaps also to changes in the cerebrum. The projection of the pain to the surface is analogous to the psychic projection of pain and other sensations beyond the stump of an amputated limb.

To my knowledge but two tumors involving the Gasserian ganglion have thus far been reported: one by Hagelstam[1] and one by Trénel.[2] In Hagelstam's case, which came to autopsy, there was present a tumor, an endothelioma, of about the size of a walnut, situated in the left, middle cerebral fossa. The dura mater and Gasserian ganglion were completely involved in the growth, which had in part perforated the underlying bone and penetrated into the posterior portion of the nasopharyngeal cavity. During life the patient had complained of constant pain over the entire left half of the face and over this area and for some centimetres upward on the hairy scalp, sensibility both for pain and touch was lost except in the area of distribution of the great auricular nerve. Over the cornea and the conjunctiva the sensibility was diminished. The sense of taste on the anterior third of the tongue to the left of the median line was lost for bitter and acid substances and for salt and sugar. The temporal muscle appears to have been atrophied. No muscular contractions could be elicited by the strongest currents. The masseter muscle felt firm and hard, but could not be made to contract to the faradic current, and the jaw could only be separated for one centimetre.

[1] Hagelstam: Deutsche Zeitsch. f. Nervenheilkunde, vol. xiii., Nos. 3 and 4, p. 205.
[2] Trénel: Bulletins et Mém. de la Soc. Anatomique de Paris, March, 1899, p. 326.

In Trénel's case there was present an angiolithic sarcoma. It had involved and penetrated the peripheral portions of the right Gasserian ganglion. On microscopic examination, the latter, however, still presented nerve-cells of normal aspect. During life there had been present profound hypæsthesia if not complete anæsthesia of the right side of the face. The patient also presented marked mental depression, was difficult to examine, and the limits of the sensory loss could not be accurately determined. No atrophic nor circulatory disturbances were noted in the eye.

Two other cases have been reported in which the Gasserian ganglion, though not directly involved, suffered from the pressure of a tumor. In one of these, reported by Krause,[1] there was a large cholesteatoma which filled the third ventricle and also involved the adjacent parts, such as the chiasm and sella turcica. The tumor was found to be entirely within the dural sac and there was no involvement of the Gasserian ganglion whatever. The patient had suffered for sixteen years from a persistent and most terrible trigeminal neuralgia. There had never been present headache, giddiness, vomiting, irregularity of the pulse nor any other cerebral symptoms. An examination of the eyes by one of the most distinguished ophthalmologists had also failed to reveal any abnormalities. As he regarded the symptoms sufficient to establish the intracranial seat of the affection, Krause decided to remove the Gasserian ganglion, although peripheral nerve operations had not yet been attempted. The removal was followed by complete relief of pain. However, about two weeks after the operation, headache made its appearance and cerebro-spinal fluid began to trickle from the point of drainage. Notwithstanding the insertion of a drainage-tube and other surgical measures, fever set in and the patient died about four weeks after the operation. It is remarkable that in Krause's case nothing could be discovered of the tumor at the time of the operation, but this is no doubt to be explained by the fact of the complete intradural situation of the growth. Krause explains the neuralgia from which the patient suffered by compression of the Gasserian ganglion and its roots. He also considered it probable that the

[1] Krause: Die Neuralgie des Trigeminus, p. 101.

tumor began at the upper edge of the petrous bone; this bone itself was entirely normal.

Another case was reported by Homen,[1] in which a tumor, an endothelioma of the dura mater, had pressed upon and flattened the Gasserian ganglion and its branches. In this case there had been present toothache in the left upper jaw, intense pain and sensations of cold in the entire left half of the face, and complete anæsthesia, the latter also involving the forehead, the anterior part of the parietal and the temporal regions. The conjunctiva was entirely without sensation, much injection, somewhat swollen, and the reflexes were absent. The cornea was cloudy, with ulceration on its inner edge. The mucous membrane of the nose and of the left half of the tongue was anæsthetic. The patient appeared to be deaf in the left ear. In addition the entire left half of the face was sunken and atrophic and the left angle of the mouth somewhat depressed. In other words, there was in Homen's case, in addition to the neuralgia and anæsthesia, a hemifacial atrophy. Although the ganglion itself was not involved in the growth, a microscopic examination of the peripheral portions of the trigeminal nerve revealed a far-advanced degenerative atrophy of all its branches. This was also the case with the roots of the trigeminal nerve.

Dr. Keen's Surgical Report.

I first saw Mr. X., with Dr. Dercum, on November 23, 1899, and learned his earlier history. The case at once impressed me as different from an ordinary tic douloureux. As my notes taken at the time state, the pain was not that paroxysmal darting one we see in tic, but a severe and constant aching pain. In view of the removal of the submaxillary endothelioma by Dr. Abbe, and of a later resection of the infraorbital nerve, without any relief, I suspected that there was probably an endothelioma within the skull, involving the ganglion, and that any extracranial operation would be useless. After talking the matter over with Dr. Dercum, we decided on operation on the ganglion.

First Operation. November 27, 1899, the usual Hartley-Krause

[1] Homen: Neurologisches Centralbl., 1890, p. 385.

operation was done. As soon as the dura was exposed it seemed to be more tense than usual. When I separated it from the middle fossa of the skull, as I approached the median line, I found that it became extremely adherent, much more so than I had ever before observed. As a result of this it was extremely difficult to find the foramina rotundum et ovale. As soon as I reached the vicinity of the ganglion I appreciated that there was a hard, not very irregular, mass of considerable size occupying approximately the site of the ganglion. The dura, when lifted from the base of the skull, could be rolled over a mass about as thick as the forefinger, and I was now thoroughly convinced that we had to deal with a new growth. After much trouble I finally found the seat of the foramen rotundum, but instead of the usually large opening, there were two small depressions in the bone, separated by a bony bridge. Whether they were complete foramina could not be determined, as no probe would pass through these small openings. No trace of the second division passing through these minute foramina could be found. The foramen ovale, however, was found and the third division passing through it, though both the foramen and the nerve were smaller than usual. Partly with the scissors, partly with the gouge, and partly with the Allis dissector, I was able to remove a mass estimated to measure 3.5 by 1.5 cm. I was not able to remove it as an entire mass but piecemeal. It extended from the cavernous sinus, which, as far as I could judge, formed its inner extremity, to the outer extremity of the foramen ovale. It stopped short of the foramen spinosum, for the middle meningeal was found pulsating and was tied, lest in the removal of the growth I might possibly divide it and be embarrassed by serious hemorrhage. The removal of the mass involved a large opening in the dura, through which a considerable amount of cerebrospinal fluid escaped. I was then confronted with this problem: in order to remove the inner portion of the tumor, it would be necessary deliberately to open the cavernous sinus. After consultation with Drs. Taylor, Dercum and Spiller, I decided not to do so, on these grounds: If the tumor were malignant, there were unquestionably other remnants left elsewhere by which it would be reproduced, and the removal of the wall of the sinus would be useless, and if it were not malignant, the removal of the major portion of the tumor

would probably be followed by the disappearance of the remainder. Moreover, the removal of the outer wall of the sinus would have involved, in all probability, destruction of the third and fourth nerves themselves, thus producing an extensive ophthalmoplegia, and, in addition to this, there would have been also a possibility of injury of the sixth nerve and of the carotid artery. The fragments removed were handed to Dr. Spiller. The osteoplastic flap was replaced and the wound closed, a small gauze wick being passed under the posterior superior angle between the bone and the dura, as a drain. The operation lasted nearly two and a half hours. He was placed in bed in a very satisfactory condition.

From the surgical point of view his physical recovery was all that could be desired. The day following the operation his temperature rose to 100° F. With that exception it was never above 99.4° F. The stitches were all removed on the sixth day, the wound being entirely healed. On the third day I removed the stitches by which the eyelids had been closed at the time of operation, and after careful disinfection I placed a Buller shield over the eye. This was removed night and morning and the eye washed with warm boric-acid solution.

No ocular disturbances followed the operation. The Buller shield was removed after twelve days. The patient left the hospital on the ninth day after operation.

From the mental stand-point, however, things were quite different. The operation was followed by a traumatic insanity, amounting almost to acute delirium, so that the patient had to be watched very carefully lest he do himself or others harm. A part of this I attributed to the withdrawal of his morphine, which was rapidly reduced, and by the sixth day was cut off entirely. Various hypnotics were tried and did some good. No reliable examination to determine the sensibility of his face could be made at this time. He, however, complained constantly and bitterly of pain in the head and the left side of his face, especially in the upper teeth. His mental condition, however, gradually improved, and by about December 12th, the fifteenth day, he was fairly clear. Another complication also was a great deal of vesical pain, the result of stricture, which I dilated with great benefit. As the pain steadily continued, and, according

to his assertion, was even worse than before, after consulting with Dr. Abbe it was determined to operate again.

Second Operation. December 26, 1899, Dr. Abbe was kindly present, and, with Dr. Taylor, assisted me in the operation. The former osteoplastic flap was turned down. The bony portion of the flap was in a few minutes caught by a gauze sponge, and with but little force was torn loose from the flap. I did not replace it. The dura was then lifted up, as at the first operation, and without serious hemorrhage. Dr. Abbe confirmed my observations as to the existence of the foramen ovale and the blocking up of the foramen rotundum. Between the two we found a third opening in the base of the skull, which we judged to be the result of the gouging of the first operation. A moderate blood clot occupied the site of the former operation and a considerably thickened mass of tissue corresponding to the old tumor could be felt. We then decided to open the dura and attack the tumor from within. This was opened by an incision a little smaller than that in the bone. As soon as the flap of dura was turned down, I noticed on the inner surface of the dura quite a number of small granulations like miliary tubercles. They were about 1 to 2 mm. in diameter, and were apparently clear and translucent. There were one or two doubtful little granulations on the pia, but on the flap of dura turned down there were, I judge, forty or fifty sprinkled over it. Later some others were seen on the dura lining the base of the skull, but much less numerous than those on the flap. Three of these little granulations were excised and given to Dr. Spiller. The pia was a little clouded along the vessels, but elsewhere was perfectly clear. The brain was now lifted up by a broad brain retractor and a very satisfactory view of the base of the skull obtained. As soon as the site of the Gasserian ganglion was disclosed I at once saw the opening in the dura, as large as the end of the thumb, caused by the first operation. It was partly filled up by a translucent gelatinous mass which resembled, to the eye, a sarcoma having considerable consistency. Along the side of it was a considerable mass of blood clot and débris. All of this was finally gouged out, and there was left a cavity so large that an ordinary, round, gauze sponge was easily packed into it. The site of this cavity was the inner end of the

petrous bone. The carotid was not seen. The ridge along which runs the superior petrosal sinus had been destroyed by the tumor. I then sought for the pons, and, on lifting the brain a little further, there came very readily into view the opening of the tentorium and the pons at a lower level. A considerable amount of cerebro-spinal fluid welled up from the spinal canal and mingled with the small amount of blood which escaped. This required constant sponging to keep the operation field clear, but there was no difficulty in determining the facts. The temporosphenoidal lobe was slightly lacerated by my retractor, but not to any notable extent. Having apparently done all that was possible, the dura was closed with continuous catgut suture and the external wound with interrupted silkworm-gut suture. A small extradural gauze wick was introduced as a drain and to arrest the hemorrhage from a small bloodvessel on the surface of the dura. The operation lasted an hour and a quarter, and he suffered very little from the shock. The temperature after the second operation fluctuated a little above and a little below 100° F. The wound healed by first intention, except at the point of drainage, where a little blood and then bloody serum escaped for about ten days. The patient went to his home in Louisville on January 9th, the wound being entirely healed.

Following this operation, after two or three days, during which he was rather dull and stupid, there was also very considerable traumatic insanity. This delirium, however, was not as prolonged nor as severe as that which followed the first operation. He had recovered almost entirely from it within a week.

Dr. Walter J. Freeman examined his ears, as he had suffered from aural discharge for a long time, and reported that the left membrane was retracted and scarred, the diminution in the degree of hearing being exaggerated by the collapsed membrane. On the right the membrane was absent and there was free discharge. He has been treated for the discharge from his ears on and off since he was a child. The doctor reported as follows: "I do not know what efforts have been made to stop the discharge, but where it is long continued I would rather suspect involvement of the bone. The only thing which promises relief is to open the mastoid freely down to the middle ear and remove the diseased bone. I do not, however, think

there is any need for operation in his case at present. His hearing was benefited by inflation, but there was no marked increase. Indeed, the condition of his ears is too bad for us to look for any great benefit in this direction.

REMARKS.—The present case is the first case of tumor of the Gasserian ganglion reported in the United States, and is, so far as I can learn, the third tumor ever reported. It is the first case that has ever been operated on. The operative technique was very satisfactory and his recovery from both operations of so serious a character most gratifying. The view of the base of the brain at the second operation was the most extensive I have ever seen.

DR. SPILLER'S PATHOLOGIC REPORT.

(From the William Pepper Laboratory of Clinical Medicine. Phœbe A. Hearst Foundation.)

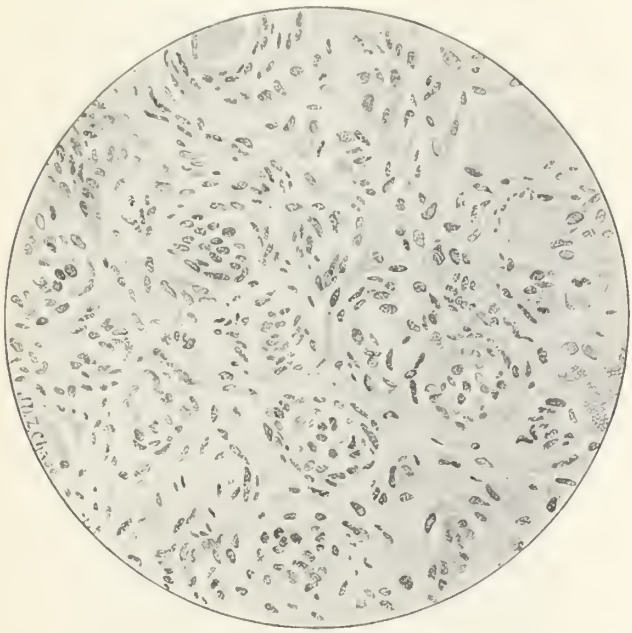
The tumor consists of cells forming long columns, or more irregularly-shaped masses, and at some parts a plexiform arrangement of these cells is seen. In some places the tumor cells are very numerous and the nuclei are small and round, so that the growth resembles a round-cell sarcoma in these portions. The cells which form the long columns have chiefly elongated or oval nuclei, and these stain deeply, but at some parts of the tumor the columns are formed of cells with large oval nuclei which take the stain less intensely. The tumor is, in places, rich in fibrous tissue, and this latter appears to consist in part of empty nerve-sheaths, the nuclei of which have undergone great proliferation. Many of these nuclei are long and narrow, although some are oval. The tumor is not very vascular.

The Gasserian ganglion is invaded by the new growth, although some parts of the ganglion are relatively free from tumor cells. The nerve-cells within the ganglion are not so numerous as in normal ganglia, and this decrease in number is more noticeable at some parts than at others. Some of the nerve-cells are more shrivelled than can be explained by the method of hardening (Müller's fluid), and the nuclei of the capsules about some of the nerve-cells are unusually numerous—indeed, in some parts the nerve-cells seem to have disappeared while the cells of the capsules have proliferated and filled the spaces left by the destruction of the nerve-cells. (See Fig. 3.) This

explanation is suggested by the concentric arrangement of nuclei seen in certain parts of the ganglion.

In certain parts of the tumor a few medullated nerve-fibres are found, but these are greatly degenerated, and empty nerve-sheaths are more numerous than medullated nerve-fibres. When these apparently medullated nerve-fibres are examined more carefully they

FIG. 3.



Oc. 3; Ob. 7. A portion of the Gasserian ganglion in which the nerve-cells have almost entirely disappeared. A concentric arrangement of the nuclei may be seen in different parts of the field, seeming to indicate that the cells of the capsules have proliferated and filled the spaces left by the destruction of the nerve-cells.

are seen to consist of separate blocks of myelin, and are, therefore, greatly degenerated. Professor Keen was able to positively determine the third division of the ganglion, and this division was kept separate from the pieces of tumor removed. Most of the nerve-fibres in this division are destroyed. (See Fig. 4.) Within the tissue known by the presence of nerve-cells to be a part of the ganglion a medullated nerve-fibre can only occasionally be found, and even

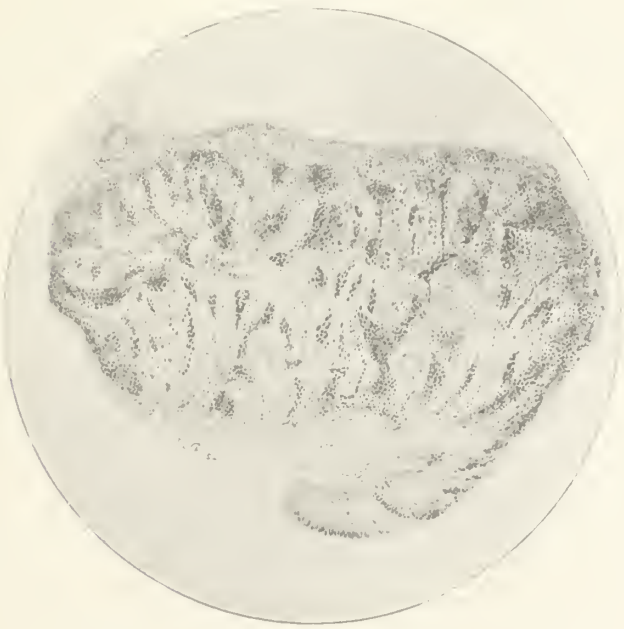
FIG. 1.

The third division of the Gasserian ganglion almost completely degenerated. The black dots and lines indicate the few normal nerve-fibres remaining.

these are much degenerated. As this portion of the tissue was hardened in Müller's fluid alone, without the addition of formalin, the failure to detect numerous medullated fibres cannot be explained by the technique employed.

At the time of the second operation small nodules a little larger than the head of a small pin were found on the inner side of the dura, somewhat resembling miliary tuberculous growths. These are

FIG. 5.



One of the miliary endotheliomas on the inner surface of the cerebral dura.

slightly larger than the tubercles usually seen in cases of miliary tuberculosis of the brain, and they grew from the inner surface of the dura and were not in the cerebral pia along the bloodvessels, where miliary tuberculous growths are more commonly found. These minute nodules of the dura seemed to me to be related to the tumor of the Gasserian ganglion, although they were *on the inner side of the dura* and in no direct connection with the large tumor mass. I expected to find that under the microscope they would have the

same structure as the large tumor of the ganglion. This proved to be the case, as one of these minute nodules examined is unquestionably an endothelioma well defined from the surrounding tissue of the dura. (See Fig. 5.) A small piece of dura removed with this nodule was also studied. Its inner surface showed accumulations of cells, some with round and some with oval nuclei, and two of these accumulations examined were separated from one another by nearly normal dural tissue.

The microscopic study of the tissue removed by Professor Keen shows that the growth is an endothelioma invading the Gasserian ganglion, destroying many of its nerve-cells and causing almost complete degeneration of the medullary sheaths of the nerve-fibres.

The tumor is unquestionably an endothelioma, although in some parts the structure is not entirely typical of this form of growth. Endothelioma, according to Birch-Hirschfeld,[1] belongs undoubtedly to the connective-tissue tumors, and in some cases a sharp distinction cannot be made between it and sarcoma. When the formation of endothelial cells occurs with proliferation of the stroma, it is doubtful, he thinks, whether such a tumor can be classed with the endotheliomas, and it should more properly be called an endothel-sarcoma; when the fibrous tissue is excessive it may be called endothelioma fibrosum.

Ziegler[2] says that the alveolar, tubular or plexiform arrangement of the endothelioma is very distinct only in the early stage of tumor formation, and that in later stages the proliferation of connective-tissue cells makes the tumor resemble a sarcoma. Endothelioma cannot, therefore, be sharply separated from sarcoma.

The tissue removed from the patient is so typical in many places of endothelioma that the growth may be called an endothelioma, although at parts considerable accumulations of round nuclei are found, and at other parts much fibrous tissue consisting partly of empty nerve-sheaths. In the small nodule removed from the dura the typical structure of endothelioma is seen.

[1] Birch-Hirschfeld: Lehrbuch der Path. Anatomie.

[2] Ziegler: Lehrbuch der Allgemeinen Pathologie. Ninth edition, vol. i. p. 431.

The proliferation in some parts of the ganglion of the nuclei of the cells in the capsules about the nerve-cells is an interesting observation. We might expect these cells to proliferate in an endothelioma of the ganglion. The capsules of the nerve-cells of the spinal ganglion, according to v. Lenhossék[1]—and the same is true of the cells of the Gasserian ganglion—consists of connective tissue which passes into the sheath of Henle of the axis-cylinder process. This capsule, especially about the larger cells, presents a laminated structure consisting of two or three layers and containing small flat or elongated nuclei. The inner surface of the capsule is lined with one layer of epithelium. In many animals the cells are flattened like endothelial cells, but in man they are quite large and rich in protoplasm and the nuclei are large and round or elliptical. Von Lenhossék at one time believed that a lymph space existed about the cells of the spinal ganglion, but in his study of ganglia removed from an executed man soon after death and hardened rapidly, he found that a pericellular space does not exist. Whether the cells lining the cell capsule are to be regarded as endothelial or not seems undetermined. Their proliferation in an endothelioma might occur whether the cells resemble endothelium or epithelium.

The great degeneration of nerve-fibres of the fifth nerve and the pressure caused by the tumor explain the intense pain felt by the patient, but it seems surprising that objective sensation in the face could have been so well preserved when the medullated fibres were so greatly degenerated. Axis-cylinders deprived of medullary sheaths may have existed, although such naked axis-cylinders are always difficult to detect.

In two excellent papers on endothelioma that have recently appeared (Kelly,[2] Sailer[3]), in which the literature is carefully considered, no mention is made of miliary endothelioma, and Sailer says that endothelioma of the dura is almost invariably benign and rarely gives rise even to pressure symptoms. Dr. Kelly and Dr. Sailer

[1] v. Lenhossék, Arch. f. Psychiatrie, vol. xxix., No. 2, p. 315.
[2] Kelly: The Philadelphia Monthly Medical Journal, February, 1899.
[3] Sailer: Contributions from the William Pepper Laboratory of Clinical Medicine.

inform me that in their study of the literature of endothelioma they have found no mention of the occurrence of numerous small endotheliomas. Our case of tumor of the Gasserian ganglion caused what may be regarded as numerous metastatic growths and—even more uncommon—miliary endotheliomas. I have also had a case of endothelioma of the cerebral dura in which pressure symptoms were very marked.

SURGERY OF THE FIFTH NERVE FOR TIC DOULOUREUX.

By ROBERT ABBE, M.D.,
SURGEON TO ST. LUKE'S HOSPITAL, NEW YORK CITY.

[Read at a special meeting held April 20, 1900.]

It is the uniform experience of surgeons that there is no more fearful pain for a patient to endure than the persistent recurrence of what one of my patients always called "crashes of pain" in the side of the face. Its immediate, though transient, relief by simple section of the superficial nerve is so much like the magic of art that it has been given as wide a trial as it ever should have. Brodie's reference to the "notorious failure of nerve section" in permanently relieving tic douloureux, echoed by Velpeau, Stromeyer and others of that time, is emphasized to-day by accumulating experience. The accepted explanation of its failure, in that reunion of nerve channels is effected by natural laws of repair, has led to so many attempts to overcome this mischievous act of nature that we may now consider the success and failure of each and learn their relative value.

If the cause of pain is central, we may rightly assume that operation on the superficial nerve will not destroy the pain sense of the patient—hence it is useless. If the cause be peripheral, we must so far destroy the communicating channel as to prevent bridging over the gap by any reasonable reparative action. Hence the value of extensive resection of the nerve seems to be in direct proportion to its extent.

As the resection of the infra-orbital part of the second branch was replaced by Langenbeck's division on the floor of the orbit, and resection anterior to that, so the latter was displaced by Carnochan's more extensive resection from the foramen rotundum to the cheek.

This latter has held its own with more recent methods, and may now be fairly considered alongside of the Salzer and intracranial methods.

To speak of the Carnochan operation as "having been done" does not rightly justify the addition of many so-called cases in a statistical list, for, like other difficult and dangerous operations, it is possible to half do it by a bungling method, poor assistants, bad light, inadequate instruments, or encountering very hard, unmanageable, posterior antrum walls.

I have had a case said to have had the operation done before, where I found much of the nerve still remaining. In my own experience I have found considerable inequality in extirpation of the nerve and branches. In judging of the value of methods, all who have had a varied experience may give weight to their personal views. In every field of surgery the statistical method is no more a criterion of value than what I would call the "personal impression" method. I do not hesitate to say, therefore, that the Carnochan operation, thoroughly done, holds a high rank with the deeper and more serious operations, and does not share their gravity.

In emphasizing the value of this operation on one branch only of the fifth nerve, I have often noticed that while the patient has designated the supraorbital and lower maxillary branches as sharing the neuralgic pain, these two are, as a rule, at once and completely relieved by operation on the middle branch. Thus we can fairly ascribe much of the pain to reflex effect from some part of one branch to its neighbors. There is also a pain sense, or pain memory, as we may call it, shown in some cases of aggravated and inveterate tic which lingers for a few days in occasional cases, no matter which method of operation has been adopted. Although the majority of operations give instant relief, the patient may complain of the old pain for as long as a week afterward, and then it leaves, and he remains cured. This exhibition of persistence of pain I have seen oftener in those who had resorted to morphine. As a rule, the morphine habit engendered by tic is entirely dissipated by operation.

My personal experience in operations on the fifth nerve's branches, excluding all those on the inferior dental and on the supra-orbital, includes somewhat more than twenty cases; but of that number only

I have notes: intracranial (Hartley's method), 5; Salzer, 4; Carnochan, 11.

Seven patients were sixty years or more, three between fifty and sixty, three were thirty-two years and seven from thirty-two to fifty years of age.

There was but one death, that of a woman sixty-three years old, with severe tic. She had borne the Hartley operation well, with section and evulsion of the second and third branches. When I was about to close the wound the patient made two feeble attempts to vomit, and breathing and heart action ceased simultaneously. Every effort at stimulating and artificial respiration, even with the Fell-O'Dwyer respirator, failed to resuscitate her.

In reviewing the five intracranial cases I find most gratifying material to report.

The first patient was a woman sixty years of age, who had been subjected to a Salzer operation two years before, and had recurrence in three months. The intracranial resection was done with much ease. The second and third branches were seized with forceps and evulsed from the ganglion after section at the foramina and forcing out the stump. The patient was freed from pain and left the hospital on the eleventh day. Two months later she died of pneumonia.

The second case was a most aggravated one of eleven years, in a woman sixty-five years of age. I operated on it in 1895, by Hartley's method, evulsing the second and third nerves from the ganglion. The woman was at once relieved and remains absolutely free from pain and in good health, approaching five years from the operation (letter received yesterday).

The third patient was a frail woman who had suffered for two years from severe trifacial neuralgia extending even to the neck; the operation was done after the usual Hartley method, and this is the case spoken of previously as a death under anæsthesia from fatty heart or shock.

The fourth, a man, aged forty-six years, had become distracted almost to suicide by inveterate trifacial neuralgia of three years' standing. In July, 1896, I resected the intracranial portion of the second and third branches, between the ganglion and foramina; the

operation was suspended on the first day, on account of grave hemorrhage on every side, while exposing the ganglion. The packing was removed the second day and dissection continued ; again profuse bleeding and packing of the wound. At the third attempt excellent exposure of the nerves was obtained, and each being grasped by a broad clamp was cut off and then evulsed from the ganglion, only half an inch being torn out, but with profuse bleeding, which again required packing. When the hemorrhage was controlled, an attempt was made to curette away the Gasserian ganglion, but again bleeding forbade it.

NEW METHOD OF OPERATING.

Fearing that the nearness of the nerve stumps in the canals to the torn Gasserian might allow restoration of function and pain, I interposed a piece of sterilized gutta-percha tissue, more than one

Violent tic douloureux—three years. Hartley operation ; second and third branches excised; rubber tissue interposed. Remains well after four years. Dotted surface shows anæsthesia : vertical lines, partial anæsthesia.

inch long and half an inch wide, which more than covered both foramina. This I pressed down by an iodoform-gauze strip, which was continued so as to compress all bleeding. In twenty-four hours—fourth day after initial operation—the gauze was removed, the rub-

ber was seen to be pressed flat on the bone and the brain allowed to settle down on it. Primary union occurred, and now, after four years, the patient remains in perfect health, retaining the rubber as left at operation. The anæsthesia in his case is shown in the illustration, from a photograph taken some months afterward, when reporting his case (Surgical Society, October, 1896).

The fifth intracranial case is also one of the same class, but in a woman aged sixty-one years, who had suffered in the gravest way for twenty-four years. All trigeminal branches were affected, and there was tenderness on pressure over the external foramina, with constantly repeated shocks of pain. I did the intracranial operation in July, 1897, and resected only the portions of the second and third divisions, between the foramina and the ganglion. I made no attempt to evulse or disturb the ganglion. The hemorrhage was slight, and I carefully interposed a sterilized gutta-percha tissue layer between the Gasserian and bone, so as to more than cover both openings. Primary union of the wound and instant and permanent relief followed. At this date—nearly three years after—I have examined her and find perfect health and no trace of pain. Anæsthesia remains as after operation.

Thus, in the five intracranial cases, three remain cured, after the lapse of three, four, and five years.

I have operated on four cases after the Salzer method.

The first case was a woman, sixty years of age, operated on in June, 1893, with most satisfactory resection of portions of the second and third branches at the foramina rotundum and ovale. Recurrence of pain occurred in three months, and she was subjected to the intracranial method.

The second patient, a woman, aged fifty years, had been suffering for five years, and was relieved for one and a half years after my operation. Recurrence was in a moderate degree only, and I have never cared to subject her to the intracranial operation, on account of her general weakness and digestive disturbance.

The third case was in a lawyer, aged thirty-two years, who had suffered for ten years and had three operations on his inferior branch, by Halsted and Hunter Maguire, with relief a few months only after each. I did a resection of the second and third branches

at the foramina rotundum and ovale, by a modified Salzer operation, two years since, with instant and permanent relief. I interposed a piece of sterile rubber tissue also in this case, between the resected ends of nerve in the sphenomaxillary fossa, and it remains there after two years.

The fourth case was in a man, aged fifty-two years, who had been suffering five years on the right side. A modified Salzer operation was done, with clean excision of a portion of the second and third branches at their exit from the skull. There was immediate relief, and he has had not a twinge of pain since then, two years after the operation.

By the Salzer method there were no deaths. By the modified Salzer, adopted in two cases, I found it easy to expose the nerves in the sphenomaxillary fossa, by splitting the temporal muscle fibres vertically, without cutting the coronoid process, simply dividing and depressing the zygoma When stretched by retractors, ample space was afforded, and the coronoid could be lowered by opening the mouth at the same time. Thus the partial ankylosis following the usual Salzer method was avoided.

THE CARNOCHAN OPERATION.

This has been rather a favorite with me and represents 11 of the 20 severe cases operated on. Two patients were thirty-two years of age and 4 about sixty years of age; the others ranged between. I have been able to follow 8 of the 11 cases; 1 remains cured after twelve years; 1 after ten years; 1 died after six years, cured; 1 remains cured at five years; 2 remain well after one year; 1 was not relieved even by subsequent operation by Dr. Keen (case reported of Gasserian ganglion); 1 remained well at two years, when he was lost sight of; 3 were relieved and lost to view.

The possibility of thorough removal of the nerve behind the Meckel ganglion is well shown in the specimen passed around, where the two filaments going to that ganglion are shown.

I have been interested in noticing cases of previous operation for removal of a large branch, that the foramen of exit closes when of no further use. Thus the mental foramen soon wastes to the size of a pin-point after the removal of the inferior dental nerve.

The anæsthesia following Hartley's, Salzer's, and Carnochan's operations is interesting. The removal of the second and third branches inside the skull leaves the half nose, cheek, and upper lip only numb, but not completely anæsthetic, as is the rest of the face, owing to a nasal branch of the ophthalmic, which covers the same distribution as the orbital branch of the superior maxillary, which comes off between the external and internal foramina. When the Salzer incision is made, this branch escapes and the nose, cheek, and upper lip are often fully sensitive.

In conclusion, my experience leads me to advocate a thoroughly done Carnochan operation, with clean resection of the second branch to the foramen rotundum, for most bad cases, even when the first and third branches seem to share the neuralgic shocks. The latter are usually relieved by operation on the middle branch. If one chooses to do the Salzer, which has some advantages in being less severe than the intracranial and more thorough than the Carnochan method, then I advise a section of the zygoma turned down with skin, and a muscle splitting of the temporal rather than coronoid section. If the intracranial method is adopted, as it must be in many grave cases, I advocate the simple section and limited excision of the second and third branches from the Gasserian to the foramina, and interposition of a piece of sterile rubber tissue, impermeable and non-conducting, adequate to cover both openings. I see no reason for believing that the resection of the Gasserian ganglion is necessary to the thorough severance of nerve connection with the brain. This step may be the greatest element of danger, and, unless a tumor of the ganglion exists, is uncalled for. If a tumor exists it seems useless. In most cases of inveterate tic a chronic neuritis exists, usually of the middle branch. Hence, the quick and permanent cures which we are able to record by excisions anterior to the Gasserian ganglion.

ANGIONEUROTIC ŒDEMA OF THE SALIVARY GLANDS.

By JAMES ELY TALLEY, M.D.

[Read May 2, 1900.]

Mrs. A., aged thirty-four years, Irish-American, has been under my observation for five years. During that time she has suffered twice from septicæmia due to retained membranes following neglected abortions. The first was probably due to a badly retroflexed uterus, the second to the same cause—for which she refused treatment—and a nephritis which dates from the first abortion.

She is apparently unusually susceptible to poisons, both microbic and medicinal, as the parenchymatous nephritis dates from the first attack of septicæmia, during which attack three douches of bichloride, 1 : 8000, produced a marked ptyalism, and two grains of quinine, on two separate occasions, produced a wide-spread and intense urticaria-like eruption with much subdermal œdema. She had had a similar experience with quinine years before.

Three years ago the patient had her first attack of general urticaria, which was hemorrhagic, and since then she has rarely been free more than a week or so at a time from hives. The rash varies from a few patches to a general profuse eruption, with here and there one that is hemorrhagic. She is always dermographic. Since the development of the nephritis, five years ago, she has had four profuse hemorrhages—two nasal and two uterine. For two years she has suffered occasionally from nocturnal asthma.

At intervals of seven years - thus antedating the nephritis and the first attack of urticaria—she has complained of spells of a peculiar swelling at the angle of the jaw, but it is only recently that I saw her in an attack and was able to determine their character. Two hours after the beginning of the attack the submaxillary and sublingual glands were enormously swollen and very tender. The parotids were apparently not so largely involved, though the lower part of these glands was also somewhat larger than normal.

These attacks come on every few months, and reappear each morning from seven to eight o'clock for several days in succession. They begin with

a "tickling in the ear and throat, a drawing of the tongue," pain and rapid swelling of the glands, which reaches its maximum in about fifteen minutes, and is immediately followed by a profuse flow of saliva, which begins with a sensation as of "many little blisters bursting under the tongue," returning to normal in from three to twenty-four hours. The sensation of many blisters bursting is interesting when one remembers that the submaxillary discharges by one and the sublingual by many duets beneath the tongue.

At first this condition was always confined to the right side, but recently both sides are involved which alarmed the patient and caused her to seek advice during an attack.

These spells are preceded by nausea, much belching and gastric distress, but no colic. As far as can be determined, neither the parents nor brothers or sisters have had any such trouble. There has been no hæmoglobinuria, nor do the attacks have any relation to the menses.

The character of the swelling, the rapidity of its development, the lack of any evidence of salivary calculi, the preceding gastric distress and periodicity, especially when occurring in one who is so subject to urticaria and so dermographic, would seem to indicate a case of angioneurotic œdema of the salivary glands.

The points of special interest are that the salivary glands are involved rather than the lips, the labia or some other common seat of election, and that the secondary paresis of the vessels producing a hyperæmia increases tremendously for a time the function of the glands, as shown by the outpouring of saliva.

ASTHENIC BULBAR PALSY.

By CHARLES W. BURR, M.D.,
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AND

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(*From the William Pepper Clinical Laboratory, Phœbe A. Hearst Foundation.*)

[Read November 7, 1900.]

We will give no historical account of asthenic bulbar palsy because Dr. Henry Campbell and Mr. Edwin Bramwell have only recently, in the summer number of *Brain*, published a careful summary of reported cases, and have made a thorough critical study of the literature. It will suffice to say that the first case was reported by Wilks in *Guy's Hospital Reports* in 1877. Wilks' patient was a stout, well-looking girl who could scarcely walk. The weakness seemed to be due rather to lethargy than to palsy. She spoke slowly, and had slight nystagmus. A month later many of the symptoms of bulbar palsy appeared, coming on rapidly in about three days. She spoke indistinctly, swallowed with difficulty, and was unable to cough. The limbs were not palsied. Soon breathing became difficult, and in a few hours she died. In the then state of knowledge it would have been proper to have expected to find organic bulbar disease. None was present. Careful macroscopical and microscopical examination revealed nothing abnormal. In 1879 Erb published a similar case, and from that time till 1886 nothing more was written upon the subject. About sixty cases have been reported, with twenty-three deaths and seventeen necropsies. These figures give a

false impression of the death-rate, and the high percentage is due to the fact that neurologists are prone to report fatal cases, and many non-fatal cases are probably not diagnosed. At first it was thought the disease affected only muscles innervated from the bulb, but in recent years it has been found to be more wide-spread in bodily distribution, and now it is usually described under the title of myasthenia gravis.

For our own work we propose to relate a case that came to necropsy, to discuss another which though resembling the first in many ways yet differs enough to make its proper classification doubtful, and to speculate a little, for the matter is still speculative, as to causation.

We are indebted to Dr. Morris J. Lewis for the early history of the first case.

A woman, aged nineteen years, and married, came to the dispensary of the Infirmary for Nervous Diseases on April 26, 1899. Her family history was negative, her personal history unimportant, except that ever since her marriage, in 1897, she had had much trouble and grief—an unwished-for pregnancy and a brutal husband. In January, 1898, an abortion, probably induced, occurred. She felt no immediate serious ill effects, but about a year later began to suffer from numbness in the right hand, and later in the left hand, legs, and back. She could feel touch, pin-pricks, and the contact of objects without difficulty. The numbness was entirely subjective. Her condition remained the same until a few weeks before she came to the dispensary. She then rapidly grew weak in the arms and legs, so that she could not easily go up or down stairs, and sometimes fell when walking on a level surface.

Examination. She walked a little stiffly and as well with eyes closed as opened. Station was good. The pupils responded to light. The arms and legs were weak. The dynamometer registered fifty in either hand. The knee-jerks were exaggerated, quick, and equal. There was no ankle clonus. The elbow-jerk was exaggerated. She said she could not feel a sharp prick on the hands or legs, and only slightly on the face. She was, however, able to pick up a small piece of paper even when blindfolded. She could distinguish between hot and cold, but said she felt them but slightly. There was a slight tremor of the hands and head. The eyelids trembled and drooped somewhat, covering about half of the pupil. The lids did not come into close apposition on shutting the eyes. She could raise the lids, but could not hold them up long. The tongue was tremulous, and when protruded turned toward the left. The respiration rate was twenty-six. The heart was normal. The sphincters were under complete control. There was slight contraction, but no reversal of the color fields. She was easily

hypnotized. She returned to the dispensary a few times, and then was lost sight of until her admission to Dr. Burr's wards at the Philadelphia Hospital on April 23, 1900.

Examination. A young woman of small frame, moderately well nourished, but somewhat pale. The head fell slightly forward upon the chest, and the eyelids drooped, partially covering the pupils. The face lacked expression. She could lift the head, but said it tired her very much to hold it up. She could not close the eyelids tightly, nor open them completely; there was but little power of movement in the frontal muscles, and she could not wrinkle the eyebrows. Her gait resembled that of a person convalescing from some serious acute disease. The movements of the arms were weak. There was no distinct palsy anywhere. Speech was slow and low-voiced—not aphasic, not paralytic, but simply weak. Mastication was slow, and she complained that eating tired her. Her whole manner and bearing showed languor and weariness, but that something more than mere neurasthenia was present was proven by the fact that the amount of weakness varied greatly in different groups of muscles, being most marked in those innervated from the bulb. The knee-jerks were large, but not spastic, and were easily exhausted. Neither ankle nor patella clonus was present. Babinski's reflex was absent; indeed, the plantar reflex was normal. There were slight but quite constant choreiform movements in the face and arms. There was no muscular atrophy of the lips, face, or tongue, and of course none in the extremities. She had difficulty in swallowing, solids seeming to stick in the throat and liquids making her cough. She still complained of numbness in the extremities, but felt touch, pain, and heat and cold well. Dr. Charles A. Oliver examined her eyes and reported: "Vision in each eye is reduced one-half to one third, that of the right being slightly improved by pin-hole. Pupils are equal in size. Irides respond to light, accommodation, and convergence. Extra-ocular movements are very much impeded, especially in outward direction, and more marked to the left. Palsy of convergence in association with a variety of ataxic movements. The eye-grounds are healthy, the fields of vision somewhat concentrically diminished. The patient is unable to close the lids, the action of the left orbicularis being more marked." Her condition remained the same until May 13th, when the difficulty in swallowing suddenly became very great, the temperature rose to 101° F., dyspnœa appeared, the pulse was rapid and weak, cyanosis and coma developed, and she died the next morning.

The necropsy was made the following day; it revealed nothing. The thoracic and abdominal organs were normal. There was a small calcified focus of tuberculosis at the apex of the left lung, and the spleen was chronically enlarged and its capsule much thickened. The kidneys and adrenals were not diseased in any way. The right ovary was cystic. The thymus gland was enlarged, but there was no other evidence of lymphatic diathesis. The uterus contained a three or four-months' old fœtus. The brain, after

hardening in formalin, weighed 1250 grammes. Neither it nor the meninges showed any change on gross examination. The cerebrum was of full size, but the pons and medulla were distinctly smaller than normal. The pons was one-third less in diameter than others which had been hardened in the same way. The spinal cord was unusually broad in the lumbar region, and after removal of the dura showed a median dorsal fissure starting at the third lumbar segment and extending to the fifth. At first sight there appeared to be a distinct bifurcation of the cord, but section revealed a persistence of the posterior median fissure extending down to the commissure. The other regions of the cord were normal.

Microscopical Examination. Serial sections of the cord, medulla, pons, and floor of the third ventricle were stained by the Marchi, Nissl, Weigert, carmine, and nuclear stain methods. In the cord no pathological changes were found. In the medulla there were distinct chromotalytic changes with swelling and displacement of the nuclei in the cells of the upper nucleus of the tenth nerve (nucleus terminis vagi). The other bulbar nuclei, including the twelfth and nucleus ambiguous, were perfectly normal. We expected possibly to find changes in the seventh and third nuclei, but they were likewise normal. Sections from the cortex revealed no changes by any of the above-named methods. Sections from all the cranial nerves were examined. By the Marchi method black dots were seen scattered here and there throughout the twelfth, tenth, eighth, and fifth nerves; but inasmuch as similar appearances are seen in healthy nerves, or at least in nerves which have performed their functions well, we attribute no significance to them. Examination of the tenth nerve stained with carmine showed an atrophy of some of the nerve fibres, giving the appearance of a sclerosis. In the tenth and twelfth nerves some swollen axis-cylinders were seen. The muscles showed no pathological changes on microscopical examination.

There are two important questions in the study of this affection: Where is the disease located, and what is its nature? Morbid anatomy gives no help in answering these questions. In the larger number of necropsies no lesion at all has been found, and in the others the changes have been slight and indefinite, similar to those in our case, and of such a nature as not to prove, scarcely to suggest, that they had anything to do with the symptoms. We are compelled, therefore, to fall back upon general physiological and pathological laws, and to draw inferences from analogous affections. The disease must of course be seated in either the muscles or the motor neurons, and if in the latter it must affect either the upper motor neuron from the cerebral cortex to the bulb or cord, or the lower neuron from the

bulb and cord to the periphery or some one part, cell body, or axon, of either. The symptoms are so predominately motor that there is no need to look for disease outside the motor apparatus. The disease has been thought to be muscular, but there is much evidence against it. In a large number of cases it has been found that on applying a tetanizing (Faradic) current to the muscles at first a brisk contraction is produced, which gradually becomes feeble, and finally ceases, to reappear if the muscle is allowed a period of rest. With the galvanic current, on the contrary, no such exhaustion of muscular contractility occurs. Now faradism causes contraction essentially by acting through the nerves, galvanism by both the nervous system and the muscle directly. Further, Dr. Farquhar Buzzard, at the suggestion of Dr. Campbell, made the following experiment: A moderate galvanic current was applied to the biceps muscle, and a contraction obtained. The muscle was then faradized until it gave no response to a strong stimulus. Then it was tired out by making the patient flex the elbow against resistance, exerted until all power of flexion was lost. On applying the same strength of galvanic current as used at first an excellent contraction was obtained. Finally, on again applying the faradic current, the muscle was found to be still irresponsive. This experiment points strongly against disease of the muscle. Again, we know of no other affection due to disease of the muscles producing analogous symptoms, whereas organic disease of the bulb is not a little similar. Sudden death, a not infrequent thing in asthenic bulbar palsy, is not a symptom of muscular disease. There are several reasons for believing that the upper motor neuron is not affected. The myasthenic reaction, the exhaustibility of the knee-jerk, the pharyngeal palsy, the dyspnœa, the cardiac palpitation, all are evidence against cerebral disease. There is but little direct post-mortem evidence in favor of the bulb as the seat of the disease. The slight changes that have been found were in it and in the cranial nerves. These changes, though almost certainly insufficient to cause the symptoms, may indicate the action of a toxin. The symptoms certainly point toward the bulb as the part first and most seriously affected. Considering everything we may say with comparative safety that the affection is one of the lower motor neurons, but whether the cell-bodies or the axons are first and

most affected cannot as yet be determined. The primary seat of disease may be in the motor muscular end-plates.

As to the nature of the disease but little is known. It looks like a toxin disease. It frequently follows some mycotic affection. It kills without visible wound. That poisons arising within or without the body may cause death without producing any discoverable lesion goes without saying. That there are diseases without a visible anatomical basis must be admitted, notwithstanding the dogma that there is never perversion of function without alteration of structure. In our case, as in a few others, we are inclined to believe that pregnancy had some causative influence. She was pregnant at the onset, and again when her disease became manifestly serious. Pregnancy may seriously disturb the normal metabolism of the body, as is shown in the kidney of pregnancy, the multiple neuritis occasionally seen, and the greater predisposition of pregnant women to certain diseases. Again, the smallness of the bulb may have had some influence. This was developmental, not secondary, not due to any acquired disease, not caused by the shrinking of an old sclerosis. What nervous elements were present were normal, but they were fewer than usual, they may have been dynamically weaker. It is possible that the ill-developed bulb was unable to withstand the stress of pregnancy.

The second case is made more difficult to understand by the presence of a singular type of anæsthesia. Sensory symptoms of any kind have been but little pronounced in the cases of asthenic bulbar palsy heretofore observed. Occasionally there is some little aching at the back of the neck and in the shoulders, with, it may be, numbness in the arms. True tactile anæsthesia has never been seen. In the case we are about to relate a very interesting form of anæsthesia was present, namely, asteriognosis, the inability to recognize objects by touch, though simple tactile sense is preserved. This symptom makes the proper classification of the case doubtful; but as it more closely resembles asthenic bulbar palsy than any other disease, we place it tentatively there. It would be interesting if in the future there should be met with cases of anæsthesia in the distribution of the sensory cranial nerves causing a condition comparable or at least analogous to the motor disease, bulbar palsy. This is a speculation, of course, but it is possible that such a condition may

sometime be discovered. Our case then would be a connecting link. The history is as follows:

A young woman, aged twenty-four years, well educated and of excellent intelligence, with good family and personal history, came to Dr. Burr, in March of this year, complaining of general neurasthenic symptoms and marked pseudo-emotionalism. She dated her illness from an attack of grippe which occurred in June, 1899. Whether this attack was one of influenza vera caused by the bacillus of Pfeiffer, or the so-called influenza nostras of unknown causation, could not be determined. At all events she never completely recovered. She was tired, languid, weak, and subject to spells of crying unaccompanied by any distressing emotional feeling. She did not regard her condition as serious, accepted no medical treatment, and grew steadily worse. A week before she came under observation her left arm began to be numb, and she had trouble in picking up small objects. A day or two later the numbness extended to the left leg, and still later to the right side. At the same time her neck felt somewhat stiff and there was severe pain in the occiput. She had slight vertigo, blurred vision, and occasional diplopia.

Examination revealed a spare, rather pale young woman. She was very emotional, bursting into tears without any cause. In the midst of her weeping she would say in the most matter-of-fact way that she did not know what she was crying about, and would go on talking about matters and things in general, paying but little attention to the lachrymal flood. Gait and station were normal; the knee jerks were normal. Anæsthesia was absent. There was no deformity of the spine, nor pain on pressure anywhere in its length. Her condition was not regarded as serious. A week later, however, she suddenly developed palsy of the right face, involving the entire side. This was not an hysterical spasm, but a true palsy. A few hours later there came on excruciating pain in both ears, which lasted throughout the night, and required morphia for its relief. When re-examined there were found a little drooping of the eyelids, a little trouble in swallowing, slightly nasal voice, weakness of the muscles of mastication on both sides, but the right facial palsy had improved. On rising she would walk well for a few minutes, but soon would stagger and be compelled to sit down. All movements of the left arm were present, but all were weak, and soon she could not move the fingers or wrist against the slightest resistance. She could not squeeze the dynamometer at all. Power in the right arm and hand was a little greater. At no time was there complete loss of power in the arms. She could always make unresisted movements, but could not even lift a spoon, could not feed herself, and even unresisted movements soon tired her so much that she could not continue them. She could not write a word, could not grasp a pen strongly enough to hold it. In bed the movements of the legs were done weakly, but even at the worst

she could stand and walk a little. The legs were never so seriously affected as the arms. She could move the tongue in all directions, but complained that the tip felt as if it had been burnt. There was no muscular wasting anywhere. The electrical reactions were normal, but unfortunately no examination was made for faradic exhaustibility. She had frequent attacks of pain in the occiput and back of the left ear, especially if she sat up in bed with the head unsupported. Palpation of the neck and head did not increase the pain at the time, but after an examination pain was apt to occur. No deformity of any kind was discoverable in the occipital region or in the cervical spine. There was no spasm of the muscles of the neck. The knee-jerks were normal, and ankle clonus was not present. Touch, pain, and temperature-sense were normal on the arms, legs, and face; but later, when the power in the arms had returned and she could grasp well, it was discovered that she could not recognize objects by touch. This test, of course, could not be made while she was unable to handle objects. Smell and taste were normal throughout the course of the disease.

Dr. John T. Carpenter examined her eyes and reported as follows: "Central vision normal; field for form and color strictly normal. No reversal of the fields. No pathological changes in the eye-grounds. Refraction error; moderate hypermetropia. No diplopia except when red glass interposed, when vertical diplopia results and is constant. Right hyperphoria 2°, which is concomitant, not paretic. Ocular movements normal, though both adduction and abduction are below par (15°—5°). The only important factor elicited was the discovery of constant right hyperphoria." The sphincters were under complete control.

On first seeing the patient I did not regard her as seriously ill, but when the facial palsy appeared, followed by the difficulty in swallowing, the nasal speech, and the great muscular weakness, I began to fear a very serious issue, and believed that there was organic disease in the bulb and upper cervical cord or in the neighboring bones. Events proved this was an error, for after a few months she improved rapidly, and now is quite well.

There is no specific in the treatment of asthenic bulbar palsy. Rest is the most important element. The patients should be put to bed; Faradism does harm; galvanism is of doubtful benefit; massage is useful. As to medicines, arsenic, in our experience, is the one drug which seems to be of benefit.

APPENDICITIS *Vs.* CYSTITIS.

By JOHN B. DEAVER, M.D.

[Read November 7, 1900.]

Miss E., aged thirty-five years, admitted to German Hospital October 5, 1900, was well until the winter of 1899, when she was sick for three days with pain in the bladder, with constant desire to urinate. Four months ago she was taken sick with cramps in the abdomen, but was sick only for a few hours. During this short illness she again suffered from frequent and painful urination. Two months ago she had another attack of abdominal cramps, accompanied by nausea, vomiting, and diarrhœa, which lasted for twenty-four hours Five days before admission to hospital she was taken suddenly ill with abdominal pain referred chiefly to left half of hypogastric region and accompanied by frequent and painful urination. This attack was not attended, however, by nausea, vomiting, or diarrhœa. Urinary examination showed the presence of leucocytes and a slight trace of albumin. None of the specimens of urine had been drawn by the catheter. The diagnosis before admission to the hospital was cystitis, and the treatment accordingly directed. I was called to see the patient, and gave the opinion that the chief source of trouble was a pelvic appendiceal abscess. Operation revealed appendiceal abscess located in the pelvis. The appendix, the tip of which with the collection of pus lie in relation with the bladder, was perforated, and the right tube and ovary were infected; the latter with the appendix were removed The peritoneal toilet included flushing the pelvis and the introduction of tubular and gauze drainage. Recovery uninterrupted

L. S., white, male, aged thirty-three years, was admitted to the German Hospital with a diagnosis of cystitis. The following history was elicited: Denies all venereal diseases; uses tobacco and malt liquors, probably to excess Three days before admission to the hospital he suddenly became unable to pass water, necessitating catheterization twice daily. Upon admission he was suffering from diarrhœa in addition to not being able to pass his water, and complained of occasional pain, which was referred to the centre and left side of the abdomen low down. Upon questioning the

patient very closely a history of what was thought to be indigestion, namely, occasional abdominal pain, with nausea and inability to take food for periods of two or three days at intervals, was elicited. After three days' stay, the patient not having improved from the medication directed by my house surgeon, Dr. Moore, the latter and myself examined him carefully, when we could make out a decided difference in the rigidity of the two sides of the abdomen low down in favor of the right side. Palpation of the right side, when continued in the direction of the pelvis, caused the patient to complain. Pressure on the left side of the lower abdomen caused pain upon the right side. Rectal examination revealed high up, and slightly to the right of the median line, an enlargement of apparent size of the point of the finger, which was tender. This mass could be identified as a pathological one. We agreed that the patient was the subject of a terminal appendicitis and that the tip of the appendix occupied the pelvis. Operation revealed an inflamed appendix which occupied the pelvis. Appendix was tied to the floor of the pelvis and to the bladder by firm adhesions; the terminal half of the appendix was covered by inflammatory exudate. Immediately upon opening the abdominal cavity many adhesions were encountered, showing evidence of a previous peritonitis.

The above cases illustrate the importance of carefully eliciting a history of each case coming under our care, of examining all cases which give history of cramp-like abdominal pain for appendicitis; that the pain of appendicitis is not by any means always referred to either the umbilical, epigastric, or appendiceal regions; that when the pain in appendicitis is referred to the left side of the abdomen low down the appendix most probably points downward or the terminal part of the organ occupies the pelvis.

The writer claims to have first called attention to the significance of left-sided pain in appendicitis indicating the pelvic direction or position of the appendix.

The additional risk in the female of inflammation of an appendix which occupies the pelvis, in the shape of the serious complication of infection of the right tube and ovary, or both, and the serious consequences of the same, is a matter of supreme moment. To avoid infection of the uterine adnexæ, and the wrecking of perhaps a precious life, means that the earliest possible operation is the safest line of treatment. Even slight infection of the ovary and tube may lay the foundation for future trouble, which the specialist aims to cure by sacrificing both tubes and ovaries, and leaving a

diseased appendix to menace the patient's life. The presence of adhesions showing a previous peritonitis, therefore former attacks of appendicitis existed in both of the cases I report. May not many of the supposed cases of cure from medicine be walking around with cob-webbed peritoneal cavities? May not a cystitis be caused by migration of pathological colon bacilli into the bladder from a near-by diseased appendix?

A REPORT OF A CASE OF RESECTION OF A LARGE PORTION OF THE ILIUM FOR CHONDROSARCOMA.

By W. JOSEPH HEARN, M.D.

[Read November 7, 1900.]

The case I report is that of a patient who was admitted to the wards of the Jefferson College Hospital on the 27th of October of this year. He was forty years of age, white, married, and weaver by occupation; birthplace Germany. Both mother and father living and well; one sister died with pulmonary tuberculosis, age unknown.

His personal history: Had enteric fever at the age of ten; no specific history.

His present trouble began in the early part of 1896, when he noticed a small, painless mass about the size of an egg in the left external iliac region just above the great trochanter. The tumor gradually increased in size. He was advised by his physician, as it was painless, to leave it alone and not seek surgical aid unless it grew very large or became painful. It increased steadily in size until the beginning of the present year, when it began to grow very rapidly. It has never been painful. The tumor is somewhat spherical and about fifteen centimetres in its longest diameter by thirteen in the shortest. Near the apex of the mass there is a sense of fluctuation; the rest of the tumor was of bony hardness. The diagnosis lay between echinococcus of the pelvic bone, similar to one reported by Viertel in a peasant woman, twenty-five years of age, or a cystic sarcoma. We decided, however, that it was a cystic sarcoma. The tumor was immovable, and seemed to spring from the medullary portion rather than from the periosteum. There could be felt on portions of the tumor hard plaques, apparently bone. We determined to remove the tumor even if a portion of the ilium had to be removed with it, but to desist should we find the pelvis too much invaded. The sacro-iliac joint was not invaded by the growth, but it extended to a level of the brim of the pelvis. The patient was apparently in good condition to bear an operation, and bore his anæsthetic well. At the suggestion of Prof. Keen, who saw the patient with

me, and who kindly assisted at the operation, a T-shape incision was made, the upper incision along the crest of the ilium. This enabled us, by pushing off the peritoneum, to explore the inner margin of the pelvis and determine upon its invasion; not being invaded, another incision over the centre of the tumor, perpendicular to the horizontal one, was made down to the tumor, which was covered by the gluteal muscles; another horizontal incision was made at the lower border of the tumor. We were thus enabled to turn back the flaps and expose the entire mass. We found it somewhat pedunculated The attachment to the bone was in the iliac fossæ. After separating the tumor as much as possible from the pedicle, a chisel and gouge now was used to cut through the bone, and thus remove the tumor with its bony attachment. This was done readily where the bone was thin in the iliac fossa, but with much more difficulty through the thick edge of the ileopubic ridge. There was little loss of blood, as the gluteal vessels were pushed aside and not divided. The patient left the table apparently in good condition, but died early next morning from shock, notwithstanding everything known was done to relieve him.

I present the tumor. It is apparently an enchondrosarcoma. The apparent cyst was a softening and mucoid changes in the tumor. In its early stage it was no doubt an enchondroma, but later took on the sarcomatous change, as other non-malignant tumors often do. I can find so little literature on enchondrosarcoma in this region that I can do but little more than report this case. I regret that I am unable to report the microscopic examination as the pathologist was unable to furnish it for this evening.

DISCUSSION.

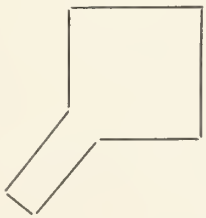
DR. W. W. KEEN: I want to say a word in reference to the case from an operative point of view, because it illustrates very well the diminution in gravity of what appeared at first to be an exceedingly difficult operation. As you see from the size, this was an enormous tumor on the buttock, and, when I examined it with Dr. Hearn, looked as though practically almost the entire innominate bone would have to be removed; but after cutting away the tumor proper by means of the chisel and gouge, it was only necessary to remove a comparatively small part of the bone.

I adopted the same technique three weeks ago in the case of a large sarcoma extending from the outer border of the breast almost to the spine, and from the fifth rib down to the eleventh. There was the same sort of mushroom-overlapping. When a large flap was made I found that

by working underneath I would be able to separate the tumor. I had then exact knowledge of the bony involvement, and found that instead of going from the fifth to the eleventh rib it was necessary to remove only portions of the fifth, sixth, seventh, and eighth ribs. The area removed was exactly the length of my hand, and exactly the width, including the partially extended thumb. Having removed the tumor I knew precisely what portion of the ribs had to be removed, and was able before opening the thoracic cavity to get underneath the ribs and so to divide each rib at two points. I was then ready to open the thoracic cavity. Had I begun with the tumor in place I would have been obliged to remove much more tissue; removing the tumor first I was able to see how much bone was diseased and to remove it. The patient has been doing very well ever since.

"A Verbal Communication on the Operation of Assaky."

Dr. Keen: I would like to present a verbal communication in connection with an amputation of the breast at my clinic to-day. The case was that of a woman who came to me a year ago with a very late carcinoma of the right breast. I did Halsted's operation, removing the breast and muscles down to the chest wall and the glands of the axilla. The woman came back with recurrence, the recurrence being in the form of nodules in the skin. I removed an area 15 by 17 cm., corresponding to the right breast, and at the same time I made a prolongation downward and outward to take in a couple of nodules just beyond.



This last incision I was able to close very readily by cross suture, leaving a large square area which it would have been impossible to cover in. In order to close it, I carried two horizontal incisions directly over to the left arm-pit, one at the lower border, the other at the upper border of the left breast. I then lifted up the entire quadrangular flap containing the breast, from the pectoral muscles, without disturbing the pectoral muscles themselves. I then drew the enormous flap toward the right, so that the left breast was drawn to the middle line of the body. Then loosening the posterior lip of the large original wound toward the back I was able to draw it forward to meet the other. Instead of there being two lateral breasts the right breast was precisely in the middle line. I covered

in the large raw surface without any trouble, except that there was a considerable amount of tension along the lower border. Between the sutures I made little nicks which widened only a little, but greatly relieved the tension. I think this will give good results with little if any sloughing of the flap.

I have never before tried this method of filling up the space left by a large mammary amputation, and it answered so well that I thought it might probably be of service to others. The plan is not original with myself, but is that advocated by Assaky (*Münch. med. Woch.*, Feb. 28, 1899).

THE NEW FORMATION OF THE FEMALE URETHRA, WITH REPORT OF A CASE.

By CHARLES P. NOBLE, M.D,
SURGEON-IN-CHIEF, KENSINGTON HOSPITAL FOR WOMEN, PHILADELPHIA.

[Read December 5, 1900.]

The entire destruction of the female urethra is of rare occurrence. But few such cases have been reported in the literature. For this reason only a few surgeons have had the opportunity of operating for the cure of this condition. Dr. Emmet has had a larger experience in dealing with such cases than any other operator. He states (*Principles and Practice of Gynecology*, 1884, p. 840) that he has succeeded in restoring the whole urethra by plastic surgery in six or seven cases, and only partially so in others. I have heard him make the statement that all of these cases were ultimately failures from one reason or another, and that it was not his intention to attempt the operation again.

In his work on *Vesicovaginal Fistula*, 1868, pp. 179, 180, and 191, is a detailed account of several cases and of the difficulties met with and the numerous operations required to make a new urethra.

Olshausen (*Ueber Urethroplastik.*, *Zeit. f. Geburts. und Gynäkol.*, Band xxxii., Heft 3) reports three cases in which he has performed the operation to restore the urethra. In two of the cases he was successful, but the third was a failure.

Baker Brown reports three cases: The first case (*British Medical Journal*, 1863, vol. ii., p. 390) was one of obliteration of the urethra and a vesicovaginal fistula. The fistula was closed and a new urethra was formed. Good union was obtained, but it was necessary for the patient to use a catheter to empty the bladder.

The second case had a vesicovaginal fistula as large as a shilling.

The urethra and neck of the bladder had sloughed away. From the description of the case it was evidently a very bad one, being complicated by a rectovaginal fistula. A new urethra was made with a trocar. He succeeded in closing the fistulæ and obtained perfect continence of urine (*Lancet*, June 20, 1863, p. 689).

The third case was one in which the urethra and neck of the bladder were entirely destroyed. This was closed by the direct operation. At first good union was obtained, but later the result was a failure. At a second operation he succeeded in effecting a cure (*British Medical Journal*, 1863, vol. ii., p. 391).

The following case has come under my own observation, and I report it as an encouragement to others to attempt the cure of similar injuries. As I operated twice upon this patient without permanent success, and by a third operation succeeded in curing her, I wish to emphasize the cause of failure by the first technique employed, and to recommend that which proved successful. This case was reported before the Section on Gynecology of the College, December 16, 1897. The history of the case and the technique of the primary operation I quote from the original report of the case:

Mrs. S., aged fifty-two years, white, American, the mother of three children, has had good health until 1896. Nothing in her history bears upon the subject of this report. At the age of forty-nine she noticed, especially when tired, a throbbing referred to the rectum. On account of the continuance of this symptom, in March, 1896, she consulted a physician in Reading, Pa., who stated that an immediate operation was required to prevent invalidism. The operation was performed and was followed by some leakage of urine, which became profuse on the removal of the stitches on the eighteenth day. The primary operation was said to be for the removal of a "blue spot" on the anterior vaginal wall. In the attempt to repair the opening in the bladder the doctor operated seven times. Four of the operations were performed without general anæsthesia. From the patient's statement it is apparent that various suture materials were employed in the different operations, including hare-lip pins; after some of them a drainage catheter was used, and after others it was not employed. All the operations failed. At this time a mass projected through the fistulous opening, which she was told was a polyp, and it was proposed to remove this. She then consulted her family physician, who told her that the supposed polyp was the prolapsed wall of the bladder. This resulted in the discharge of the doctor who had made the fistula; and, upon the advice of her family physician, a well-known gynecologist of this city was called in, who oper-

ated upon her twice in Reading and twice in a hospital in this city. Union was not secured.

Mrs. S. consulted me November 16, 1897, and on examination I found that the entire inferior wall of the urethra was gone and that a fistula existed involving the neck of the bladder. The situation of the urethra was marked by a strip of mucous membrane continuous above with the vesical wall. The opening into the bladder was large enough to admit the index finger. The edges of the fistula were cicatricial, and upon each side of the urethra extensive cicatrices were present, probably the result of incisions made at the various operations to relieve tension on the sutures. After some of the operations there must have been considerable destruction of tissue, as a large amount of cicatricial tissue was present.

The mental condition of the patient was very bad, being due, doubtless, to the failure of the eleven operations to effect a cure and to the constant annoyance of the discharge of urine. November 20th I operated upon her at the Kensington Hospital for Women. As a preliminary I procured a Sims sigmoid metallic catheter, whose diameter was one-third that of the usual catheter. The operation was performed as follows:

An incision was made along the edge of what corresponded to the original mucous membrane of the urethra from the meatus to the bladder. External to this line of incision a raw surface was made upon each side of sufficient breadth to make a firm urethral wall. The edges of the fistula into the bladder were then denuded, an effort being made in the denudation to secure as small a neck to the bladder as possible. Deep incisions were then made, parallel to the long axis of the vagina upon each side of the proposed urethra, to secure flaps out of which to form the new urethra. On the left side it was necessary to detach the soft parts entirely from the pubic bone in order to overcome tension.

The sutures were introduced in the following manner: The small catheter was introduced and held in position; over this was sutured, with a running suture of No. 1 cumol catgut, the mucous membrane of the bladder and that of the new urethra; interrupted silver wire sutures were then introduced to close the opening into the bladder and to form a new urethra. An effort was made to pass the sutures at the neck of the bladder in such a way as to catch, if possible, the muscular fibres which form the sphincter vesicæ. The silver-wire sutures were then tightened and twisted, closing the fistula in the bladder and building up a new urethral wall. To re-enforce this line of sutures, and to secure an even better approximation, a silkworm-gut suture was placed between each of the silver-wire sutures. Sutures were then passed in the direction of the axis of the vagina to close in part the incisions made at each side of the restored urethra, and, more especially, by approximating the ends of these incisions, to still further guard against tension on the restored urethra. The deep incision on the left side was then packed with gauze. The operation lasted about one hour and a quarter.

The next problem was to secure healing, and I determined to leave the catheter *in situ* until the newly-formed urethra had firmly united. After two days the catheter became blocked by a deposit of urinary salts, after which time the bladder was washed out daily with boric acid solution to overcome this difficulty. The catheter remained *in situ* until the twelfth day, after which it was removed daily for cleansing. The line of union healed by first intention throughout, although the tissue which made up the left side of the restored urethra was cicatricial in character and its vaginal aspect was bare of mucous membrane. Three weeks after the operation the deep incisions had filled up by granulation and only a small area remained for cicatrization.

The patient is able to retain her urine for from three to five hours, after which time she has not perfect control over the bladder and is apt to discharge a small quantity of urine if the bladder is not promptly evacuated.

I would like especially to call attention to two points in the technique of the operation, as I believe a successful result was dependent upon them. The first was the use of a very small catheter, which was left in position until primary union had been secured. I felt, and still feel, certain that an attempt to pass through a somewhat distorted canal daily or more frequently would result in the perforation of the canal and failure of the operation. The second point was the method used in suturing. By first restoring the mucous lining of the canal with a continuous catgut suture it was possible to insure a narrow urethra of uniform diameter, and in the subsequent restoration of the wall of the urethra it was possible to disregard the urethral canal and to consider merely the building up of a firm urethral wall. It was possible, also, to study the problem of tension more carefully. It was found necessary to make a very extensive detachment of the soft parts from the pubic arch in order to secure a flap of tissue without tension.

April 16, 1898, the following letter was published in the *American Journal of Obstetrics*, p. 622.

DEAR SIR: I desire to add the subsequent history of the case of operation for the restoration of the urethra and for the closure of a vesicovaginal fistula involving the neck of the bladder, which was reported in the March number of the *American Journal of Obstetrics*.

"This patient returned home apparently soundly healed and was able to retain urine for four hours. Shortly after her return home she wrote me that her urine was escaping constantly, and again consulted me for examination. A small fistula was found just at the internal orifice of the urethra. This undoubtedly was produced by lateral traction on the restored urethra. This traction was brought about by the healing of the lateral incisions, which were made to relax tension at the time of the operation. The expe-

rience further illustrates the intractable nature of such cases. It adds another to the cases reported by Emmet in which the line of union gave way some time after apparent cure. It is my purpose to attempt the restoration of the urethra by the formation of flaps, and I hope to report the results thus secured.

"Very respectfully yours, etc."

The fistula which formed at the internal orifice of the urethra was so small that I felt it was worth while to attempt its closure without disturbing that portion of the operation which had remained healed. On January 10, 1898, the edges of the fistula were freshened, deep lateral incisions parallel to the course of the urethra were made to separate the tissues from the pubic arch, so as to relieve tension from the line of union, and the fistula was closed. This operation was an entire failure. Not only did the fistula fail to close, but much of the urethra which had been united at the time of the operation broke down.

It was plainly evident that the cause of failure was the tension upon the line of union, and I determined to abandon the direct method of operation and to resort to the formation of a flap. The parts were permitted to heal soundly, and the general condition of the patient was improved by suitable treatment, and on June 15, 1898, I operated for the third time, making the fourteenth operation which the patient had undergone. The following points were determined upon in the performance of this operation:

1. To elongate the urethra so that the orifice of the new urethra should be at the clitoris instead of at its normal site. This was determined upon to increase the retentive power of the bladder lost by the destruction of the sphincter vesicæ.

2. The first step in the operation consisted in suturing the mucous membrane over the small sigmoid self-retaining catheter before mentioned (as in my first operation) to constitute the lining membrane of the new urethra. What was left of the anterior wall of the original urethra was employed, and between the external orifice of the urethra and the clitoris the mucous membrane of the vestibule was utilized. Two parallel longitudinal incisions were made and the mucous membrane sufficiently loosened to enable me to suture it over the catheter with a continuous catgut suture, thus forming the mucous lining of the new urethra. The raw surface over the site of the urethra was broadened by splitting and by denudation, especially in the neighborhood of the neck of the bladder, in order to secure a broad raw surface upon which to unite the flap.

3. The left labium minus was selected as the tissue from which to form the flap. This was detached from the subjacent structures quite easily (the incision beginning at the left border of the urethra), as it proved quite simple with traction and slight dissection to unfold it. The tissue of the labium minus and of the adjacent labium majus is so elastic that it was quite easy to draw it over the raw surface and well back into the vagina.

Numerous interrupted catgut sutures were used to attach the labium minus to the raw surface which had been prepared, and the edges of the labium minus were sutured with silkworm-gut sutures to the right border of the raw surface along the urethra and within the vagina. At the anterior end of the new urethra near the clitoris the lateral surfaces of the labia minora were sutured together. In this way not only was the floor of the urethra made of the tissue of the left labium minus, but also a flap of considerable thickness was secured over the region of the neck of the bladder. This was done not only in the hope of effecting a cure of the condition without further operation, but also with the intention that in case a fistula appeared at any point to prepare the way for its closure by furnishing sound tissue through which to operate.

The after-treatment was similar to that employed in the first operation. The result was all that could have been hoped for. Firm union was obtained. With the aid of a small tampon introduced just within the vagina, to make pressure upon the internal orifice of the urethra and to elevate slightly the base of the bladder, the patient was enabled to retain her urine for a number of hours during the day and to sleep soundly during the night. At this date the result is equally satisfactory. She introduces herself a small tampon daily, and with this assistance can retain her urine for hours at a time during the day and also during the night. She is thus enabled to perform her household duties in comfort and to take her place in society.

I wish to commend strongly to others who may be obliged to operate for this condition, or a similar one, the plan of utilizing the labium minus as a flap from which to secure sound tissue. Owing to the extreme elasticity of the labium minus and the labium majus, they can be employed not only to supply the floor for a new urethra, but also to supply tissue for the closure of extensive fistulæ involving the base of the bladder. So far as I know the utilization of a labium minus as a flap to form a new urethra is original with myself.

The experience of Emmet and the results of the six operations upon this patient, two by myself, and four by another trained operator, is a sufficient basis to advise against persistence in the ordinary direct method of operation, if this fails more than once or twice. Experience has shown so clearly that the cause of failure is the cicatrization following the healing of incisions made to relieve tension, that there can be no doubt upon this point; hence, whenever it is necessary to employ deep and extensive incisions to relieve tension in order to secure flaps for the direct operation, it is better not to attempt the direct method, but to utilize the tissue of the labium minus as a flap.

CULTIVATION OF THE ÆSTIVO-AUTUMNAL MALARIAL PARASITE IN THE MOSQUITO—ANOPHELES QUADRIMACULATA (OR CLAVIGER)—WITH PRESENTATION OF ORIGINAL SPECIMENS.

By ALBERT WOLDERT, M.D.,
OF PHILADELPHIA.

[Read by invitation, December 5, 1900.]

Mr. President and Gentlemen of the College of Physicians:

I desire to return sincere thanks to you and your President for the honor accorded me to address you this evening.

After having made numerous dissections of the fresh mosquito and comparing these with serial sections I have been able to determine the anatomy of the mosquito.[1]

This being done, the next question at hand was to find whether or not Anopheles could be found in or near Philadelphia. Previous to June 19, 1900, I had not found a single specimen of this genus, although my investigations had been carried on throughout the entire winter.

One thing seemed to be suspicious, and that was in the previous fall I had treated a case of æstivo-autumnal malarial fever[2] which had undoubtedly originated in Philadelphia, and it was in this locality where my first efforts were made to capture the adult Anopheles or their larvæ. Periodic trips were made to this section every few weeks during the past summer, which has been the warmest for the past thirty years, but I brought back only the larvæ of Culex or other insects.

However, on the fourth trip, and on the homeward journey, June 19, 1900, I dipped up a small, dark, and more or less striped mos-

quito larva which lay flat upon the surface of the water. Others were caught and raised to adults, and proved to be the Anopheles quadrimaculata and Anopheles punctipennis, both of which are capable of acting as the intermediate host for the malarial parasite. Subsequent to this trip other visits were made to the same locality every few weeks from June 19 to the latter part of October, and I never failed to catch the larvæ of the Anopheles, and in this way have succeeded in raising about two hundred adult mosquitoes from the larvæ, some of which have been kept as long as twenty-seven days. Subsequently making other journeys to regions where cases of malarial fever had developed, I learned that Anopheles could nearly always (all but one) be found, and in this way have discovered five localities within the city limits of Philadelphia where Anopheles, or malaria-carrying mosquitoes, have their breeding-grounds.

These investigations up to this period, therefore, tended to corroborate the observations of others, that "*where there is malaria, there are mosquitoes*"—Anopheles. But it does not necessarily follow that where there are mosquitoes there are cases of malarial fever. In proof of which I wish to cite that information has been furnished me that no autochthonous case of malarial fever has developed at the League Island Navy Yard (Philadelphia) for several years, although there is a breeding-ground of the Anopheles not far away. I have been able to catch the Anopheles in the Pocono Mountains of Pennsylvania and at an elevation of nearly 3000 feet above the sea level. In this region there is little malarial fever, though five miles further, or near Tobyhanna, I have been informed that the disease is somewhat prevalent, and should one look carefully in the latter locality a breeding-ground for the Anopheles will doubtless be found.

Anopheles will always remain harmless unless someone suffering with malarial fever comes among them.

The malarial parasite of man is capable of being classified into a natural order (Gymnosporidia), a class (sporozoa), into different genera (*a*. Hæmamœba, and *b*, Laverania), and into different species: (I.) hæmamœba malariæ (quartan), hæmamœba vivax (tertian), and hæmomenas præcox (remittent fever) Ross;[3] and it has been further proved that the flagellated malarial parasites are of different sexes.

These different forms require for their complete biological cycle an intermediate host, without which they doubtless perish. This being so, How then can we defend the theory that malarial fever can be due to *miasm?* Or if it can be received into the system through the medium of drinking-water, what form does it assume? All such experimental evidence has seemingly failed in this direction, and only positive evidence has been furnished that the mosquito acts as the means of distribution of the infectious agent. As stated by Ross, swampy land may not produce the malarial parasite, but may be the *carrier of it.*

Malarial fever practically disappears in midwinter, then recurs in the following spring, gradually increasing in summer and toward fall. This is explained on the ground that many patients who have had an attack of the disease the previous fall, and who, not taking sufficient amount of quinine to destroy all the parasites, have a recurrence the following spring when the Anopheles appear, and in this way perpetuate the disease. In my investigations carried on throughout the past year I believe the results prove that mosquitoes *survive the winter* in this latitude.[4]

The life cycle of the malarial parasite in man and in the mosquito is totally different. In the case of man the spore invades the red blood-corpuscles, subsequently becoming larger or more expanded, and undergoes segmentation, when the spores again invade fresh erythrocytes. In the mosquito a different process occurs, which has been explained by Marchiafava and Bignami[5] somewhat as follows: Adopting the zoölogical nomenclature to simplify matters, one finds that mature sexual forms of miero-organismal life are termed gametes, the female being known as macrogametes; the cells which produce the male elements are termed macrogametocytes, and the male elements themselves are spoken of as microgametes.

During certain periods of development of the æstivo-autumnal malarial fever and its homologues of the other species, if the blood be withdrawn from the circulation and left standing on the stage of the microscope for fifteen or twenty minutes, it has been found that some of the crescents are subsequently converted into ovoid bodies, and finally are transformed into flagellated parasites or microgametocytes. From these free flagellæ or microgametes tear them-

selves loose and penetrate certain ovoid forms or female macrogametes which had not previously developed flagellæ. By this process fecundation occurs, after which it rapidly develops flagellæ, and by means of its sharp extremities has the power of penetrating dense tissues, such as the middle intestine (stomach) of the mosquito, and on doing so after a day or two becomes converted into the zygote (eoccidium, oöcyst, or sporozoon), which about the seventh to tenth day contain the spores (sporozoites, sporoblasts, blasts) of malarial fever.

After discovering the Anopheles in the locality of Philadelphia on June 19th, I was not sure that I really had the *malarial bearer*, since Howard had not at that time identified the Anopheles quadrimaculata as being the same species as the Anopheles claviger of India and Africa.[6] However, I endeavored to determine whether or not the malarial parasite could be cultivated in the species of Anopheles found here, and desire to record the following observations:

Observation I. Date, December 29, 1899. The first case was W. F., a lad, twelve years of age, whose blood contained ovoids and crescents. After a trial lasting two hours the mosquitoes would not bite. This was my first failure, which was again repeated two days subsequently.

Observation II. Date, July, 1900. A man suffering from tertian fever was bitten by two Anopheles quadrimaculata, one of which died; the other escaped.

Observation III. Date, July 20, 1900. Mrs. J. H. W., aged thirty-six years, whose blood showed a large number of tertian parasites (double tertian), which were stained with eosin and methylene-blue, and with carbol-thionin; after a trial lasting from 8 P.M. until 11 P.M. a specimen of Anopheles punctipennis would not bite. A specimen of Culex pungens caught in the room was then tried, and bit the patient. It was kept at a temperature of 84° F., and dissected on the seventh day, but exhibited no pigmented cells (zygotes).

Observation IV. Date, July 27, 1900. The patient, J. G., aged thirty-four years, whose blood contained two groups of tertian parasites, some of which were full grown, was bitten by two Anopheles quadrimaculata or claviger, and were kept at a temperature ranging from 76° to 80° F. for seven days, but contained no pigmented cells.

Observation V. Date, August 7, 1900. M. P., a patient, aged thirty-five years, who had been suffering with a double tertian infection, and in whose blood were a large number of malarial parasites, was bitten by one Anopheles quadrimaculata about two hours before a chill, and by three others from half an hour to within a few minutes of the chill. They were kept

at a temperature ranging from 84° to 88° F. The one which bit first died on the third day, and two of the others died on the fifth day. The last one survived, and was killed on the seventh day. All the insects were dissected, but contained no zygotes.

Observation VI. Date August 29, 1900. P. S., aged sixteen years, in whose blood were a very large number of tertian parasites in different stages of development, some full grown, others very small, was bitten by three Anopheles quadrimaculata, which were afterward kept at a temperature ranging from 80° to 84° F. One of these was dissected on the fourth day, but contained no coccidia (zygotes).

Observation VII. Date, August 31, 1900. K. C., aged twenty-one years, who suffered from a double tertian infection, was bitten by two of the Anopheles which had bitten Case VI. two days previously. They were dissected on September 2d, but contained no zygotes.

Observation VIII. Date, September 24, 1900. L. N., aged twenty-three years, whose blood showed two distinct groups of tertian malarial parasites, some of which were almost full grown and others in the stage of segmentation, was bitten by three Anopheles quadrimaculata about two and a half hours before a chill. They were kept at a temperature ranging from 76° to 80° F. One died on the fourth day, and all were dissected on the fifth day, but none contained zygotes.

Observation IX. Date, October 3, 1900. S. N., aged thirteen years, who suffered from a double tertian infection, and whose blood at 5 P.M. contained full grown and actively dancing parasites, was bitten at 8.30 P.M. by two Anopheles quadrimaculata, which were subsequently kept at a temperature ranging from 80° to 86° F. One died on the third day. The other one was kept for five days. Both were dissected, but contained no pigmented cells (zygotes).

Observation X. Date, October 27, 1900. P. Y., aged twenty-seven years, a patient of the Jefferson Medical College Hospital (service of Dr. Julius Salinger), whose blood showed a few ovoid and crescent malarial parasites, was bitten by two Anopheles quadrimaculata at 9 P.M. These were kept at a temperature ranging from 76° to 84° F. for three days, and then dissected. Near the lower portion of the middle intestine of one of these mosquitoes two zygotes, or pigmented oval cells, were found.

Beside these observations I also made several trips to the houses where cases of malarial fever were being treated, and in every instance but one succeeded in catching Anopheles. I must note in this connection that in many locations the families afflicted would frequently explain to me that mosquitoes had not been troublesome, and a few denied that they had been bitten. In nearly all these instances, after a search, Anopheles were found either on the

walls or the ceiling of the sleeping-rooms. This is easily explained, since patients have often told me they could feel nothing, although at the time I could see the mosquito biting.

Other Anopheles were caught in barns in a community where malarial fever was more or less prevalent, but none of these nor those from the sleeping-rooms contained zygotes.

In all the observations above recorded malarial parasites were found either in the fresh blood or in specimens stained with eosin and methylene-blue, with carbol-thionin, or by the Romanowsky method.

Summarizing the series would be as follows: Of nine cases of malarial fever seven of them were bitten by sixteen Anopheles quadrimaculata (claviger) and one by a specimen of Culex pungens, and in these only one harbored the malarial parasite.

I can only explain the cause of my failure to find the zygotes in more of these on the ground that none of the cases contained preflagellated parasites in the blood at the time the mosquitoes bit, that the insects had never deposited their eggs,[7] or else they were not in a condition to be infected.

The malarial season was now drawing to a close, and after fourteen months' work, as good fortune would have it, the following interesting case, which is the last of the series, was placed at my disposal by Dr. Frederick A. Packard, visiting physician to the Pennsylvania Hospital:

Observation XI. Date, November 1, 1900. A. De B., aged fifty years, a patient of the Pennsylvania Hospital, and who had been a resident of the United States for thirty-six years.

Family History. His father had died of old age and mother of typhoid fever.

Previous History. The patient stated that he had been sickly during youth. When twenty-five years of age he suffered from enteric fever, and subsequently remained well for a period of ten to twelve years, when he had an attack of hard shaking chills followed by fever, and lasting irregularly all the summer. At this time he was living on George's Island, New York, about four miles south of Peekskill. His residence here was adjacent to pools of water and swampy land. Two years later, while living in Croton, New York, near the Hudson River, he suffered from another attack of hard shaking chills. His residence was situated about two hundred yards south of the New York Central and Hudson River Railroad Depot and near a

brickyard in which there was standing water. (The exact spot is indicated here, hoping that some day Anopheles may be discovered in that locality.)

The third attack of chills and fever came on about six years ago, while he was living in Sumterville, Florida. His residence was about three miles east of the depot of the Florida Central Railroad, and his home was adjacent to a swamp, but mosquitoes were not troublesome.

Four years ago he suffered from a mild attack of chills and fever, and two years later had another attack. While living at Sayersville, New Jersey (about one-half mile on the west side of the town), he again suffered from paroxysms of malarial fever, recurring every two days, and always followed by fever. He remained in bed for two weeks and took quinine. After getting up he continued to take quinine for a period of several weeks. The patient states that he felt well all during last winter and until the onset of warm weather of May, 1900, when he became drowsy and suffered from vague pains. At that time he was again living on George's Island, State of New York, where the first attack of chills occurred. He states that previous to this illness he was bitten by a number of mosquitoes. At this time he drank water from a shallow spring which was poorly drained.

History of Present Illness. About the last week in July, 1900, and while still living at the above address, he began to suffer from dizziness, drowsiness, and vague pains. The bowels had been regular and the urine seemed normal. Two days after the onset of the initial symptoms he had a hard shaking chill at 2 P.M., which lasted two hours, and was followed by fever and profuse perspiration. Two days later he had another hard shiver at 1 P.M., which was followed by fever. His third chill occurred at 12 o'clock noon two days afterward, and the fourth chill at 11 A.M. two days later. He was then sent to a hospital in Peekskill, where he was given quinine for a few days, and returned to work, continuing the quinine for a week or two.

On October 15th he suddenly became sick at 2 P.M., at which time he had a hard shaking chill, lasting two hours, and followed by nausea and vomiting. Another chill followed the next day at 2 P.M. After the latter chill he had no recurrences for five or six days, when they again occurred every day for three successive days at 11 A.M., and he was admitted to the Pennsylvania Hospital on October 31, 1900.

Physical Examination, November 3d. The man had a dull countenance, and the skin and sclerotics had a somewhat yellowish tint; the tongue was moist and showed a thin whitish coat down the centre. The spleen was not palpable and the area of dulness was not increased, but it was tender in this region. The hepatic dulness appeared to be normal, though there was resistance in the region of the gall-bladder, where some tenderness was elicited. The heart and lungs were normal.

Examination of Urine, November 1st. Three successive examinations of the urine proved it to be normal.

Probable Source of Infection. While at George's Island, New York, he resided in a boarding-house at which six other people lived. None of these had suffered from malarial fever during the present year. He slept in a room with two other acquaintances. At a neighbor's house, about two hundred yards distant, certain persons had suffered from chills and fevers a few days previous to his present illness. Other neighbors had suffered with chills more or less all the year, and about the time the patient became affected the disease appeared to be more wide-spread.

About one month after this patient had the first chill one of his friends living in the same room also began to have chills and fevers, and two or three weeks later the other friend, also sleeping in the same room, began to suffer with the disease. The blood was not examined for malarial parasites in any of these cases; but if it had been, in the opinion of the speaker, the same type of parasite, it would probably have been found in all three persons afflicted, since the infectious agent was doubtless obtained from the first patient affected. A breeding-ground of the Anopheles should be expected to be found in that neighborhood.

Examination of the Blood at 7.30 P.M., November 1st. The blood was obtained from the tip of the finger and immediately examined with a $^1/_{12}$ oil-immersion lens (Leitz), and showed crescent-shaped malarial parasites and ovoid bodies, there being probably one ovoid to every three and one crescent to every five microscopic fields. Both of these varieties contained abundant reddish pigment granules arranged in a circle around the periphery of the parasite (sporozoa), and the granules were in very active motion. About ten minutes after the blood had been withdrawn from the circulation some of the ovoid bodies began to throw out toward the periphery a small rounded arm, the latter of which was actively dancing. About twenty minutes after the blood had been withdrawn a flagellated body was seen. This flagellated sporozoa had five or more arms, which it waved about in all directions. One of the flagellæ had attached to its extremity a small oval particle of a red blood-cell, which was tossed around as if it were being thrown aside.

As soon as the flagellated malarial parasite was discovered one of the Anopheles was applied, then several more, until seven had bitten. I am quite sure all the insects bit the patient, because it was easy to see them take hold, after which the blood could be seen distending the middle intestine (stomach) of the insects. At this time other blood-films were selected, and by modifying Manson's method I succeeded in staining a flagellated parasite, using the Romanowsky stain. Flagellated malarial parasites were found on the day previous to this observation and again on the following day. After biting the Anopheles were kept in a warm place for a period of about one hour, and were afterward transferred to a cupboard near a kitchen stove and kept at an average temperature of 78° F.

(Under the influence of fractional doses of calomel, followed by quinine, the patient rapidly recovered.)

Dissection of the Mosquitoes. The Anopheles had bitten on Thursday, November 1st (from 8 to 9 o'clock), and the first one was dissected on the Monday following at 10 A.M., but contained no zygotes. Two others were dissected on Monday at 4 P.M., and one of these showed the four zygotes of malarial fever presented before you, which is, I believe, the second positive result so far obtained in America, the first one having been made by Dr. W. S. Thayer, of Baltimore.

While the Anopheles only bit one time on Thursday, November 1st, between 8 and 9 P.M., and subsequently kept at a temperature averaging 78° F., and dissected on the following Monday at 4 P.M., all of the zygotes do not seem to have developed with the same degree of rapidity. As, for instance, No. 1 is a very young form, resembling the ovoid body of the fresh blood, while No. 4 is of a later period of development. Thus in this specimen many stages in the development of these pigmented cells are represented, from that of early impregnation of the female macrogamete to the more mature form of zygote. In the younger coccidium the pigment is in fine granules, while in Nos. 3 and 4 the pigment dots are considerably larger, as if there might have been a coalescence of this destroyed hæmoglobin.

The most distinguishing characteristic of the coccidium or zygote of malarial fever of the first few days, which serves as a means of identification, is the presence of reddish pigment granules similar in color to those of the malarial parasite of the blood, which is arranged around the periphery of the cell; secondly, the presence of a very faint hyaline capsule, and, third, the distinctness of this pigmented cell. Though I had not previously seen a specimen of the coccidium of the human malarial parasite, I was by these appearances able to recognize them, though a previous examination of the zygotes of proteosoma, loaned through the kindness of Dr. Ronald Ross, also helped me considerably in identifying the specimens.[8]

All of these coccidia are located in the lower portion of the middle intestine of the mosquito and near the origin of the Malpighian tubes, which for some reason appears to be the favorite site of these bodies. The other Anopheles which bit did not contain zygotes.

Description of Plate.

Macroscopic Appearance. Nothing can be determined by a macroscopic examination of the infected middle intestine of the mosquito. It still possesses a light brownish tint.

Examination with Low-power No. 3 oc., No. 3 ob. (Leitz).

With this power the coccidia cannot be seen at all. With No. 3 oc. and No. 7 lens, bodies 1, 2, 3 and 4 are seen quite clearly, the pigment dots having a brilliant reddish tint. The rest of the body of the cell has a bright hyaline tint, and in areas adjacent to the pigment appears granular. The general outline and finer characteristics cannot be determined with this power.

Examination with $^1/_{12}$ Oil Immersion, No. 3 oc. (Leitz).

Appearances of Zygote No. 1. This coccidium, as has been stated, has many characteristics of the ovoid body of the fresh blood found in cases of æstivo-autumnal fever, with the exception that in the zygote the pigment granules are slightly larger, and are pushed outward from the centre and toward the periphery of the cell, each granule being surrounded by a faint halo, which are probably vacuoles. That portion of the coccidium which contains no pigment has a delicate yellowish tint. This pigmented cell is surrounded by a very thin hyaline capsule, causing it to stand out quite prominently from the surrounding tissues. It is situated in the outer edge of the middle intestine and is somewhat superficial to the epithelial cells. It is probably eight to ten microns in diameter.

Description of Zygote No. 2. While coccidium No. 1 is situated outward and superficial to the deeper epithelial cells, this specimen seems to have selected a position among the muscular tissues which it pushes to either side. In shape it corresponds to the crescent of the æstivo-autumnal malarial fever seen in the fresh blood, thus seeming to demonstrate the power of the zygote to preserve its original type.

The pigment granules are considerably larger and fewer in number than in the crescent of the fresh blood and are arranged around the periphery. A faint colorless area surrounds each pigment granule, and very faint slit-like vacuoles are seen in several areas, but any attempt to draw them only exaggerates their appearance. The zygote is surrounded by a thin hyaline capsule, which gives it a stamped-out appearance. It is about 7μ in width and 14 to 16μ in length.

Description of Zygote No. 3. This zygote is the lowermost one, and is not far above the origin of the Malpighian tubes. It is irregularly crescentic in shape, elongated at one extremity, and is situated somewhat superficial to the softer epithelial cells and adjacent to a spiracle, as is zygote No. 1.

Here, too, the brilliant reddish pigment granules have coalesced and

have been pressed outward toward the periphery. Faint vacuoles surround each pigment granule, and slit-like clear spaces traverse the body of the cell in several localities. The central portion of the coccidium has a very faint yellowish tint. Its outline is quite sharp, and focusing up and down the appearances change somewhat, the reddish pigment becoming slightly yellow, while the vacuoles assume a different shape. This zygote is about 10μ in width and 14 to 16μ in length.

Description of Zygote No. 4. (Seen under the microscope.) This coccidium is somewhat ovoid in shape and is the largest one of all. It can be readily studied with a No. 7 lens. It, too, is located near but superficial to the deeper epithelial cells. Many irregularly shaped reddish pigment granules are arranged around the periphery of this cell, and are several times the size of the pigment found in the malarial parasite of the blood. Certain portions of the inner lining of the coccidium seem to be slightly thickened, leaving a more or less lighter area in the centre, the upper portion of which contains single pigment granules. Slit-like vacuoles extend in various directions of the body of this cell, which are brought out by focusing up and down. Certain portions of the coccidium have a faint yellowish tint, though its general appearance, as in the other coccidia, is hyaline. This cell is surrounded by a colorless rim or capsule, thus giving it a stamped-out appearance. It is about 12 to 14μ in width and 16 to 20μ in length.

NOTE.—I desire to return sincere thanks, first, to Dr. Frederick A. Packard, who has placed this case in my hands for study; next, to Prof. Simon Flexner, of the University of Pennsylvania, who has given me the facilities offered by his private laboratory for research work; also to the following named, who have given me the opportunity of studying cases of malarial fever, namely: Drs. J. C. Wilson, W. C. Hollopeter, M. T. Prendergast, J. M. Cruice, Julius Salinger, N. D. Pontius, Frank White, A. P. Francine, and to the Sisters of St. Joseph's Hospital. To Dr. Ronald Ross, of the Liverpool School of Tropical Medicine, I am greatly indebted for loaning me his specimen of mosquito infected with proteosoma, and for sending me literature on this subject, both of which have been invaluable in identifying my specimens; also to Dr. Riesman for the use of a microscope this evening; and, lastly, I must thank your President, Dr. W. W. Keen, for his very kind invitation, and to each member of the College for your polite attention.

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2. Philadelphia Medical Journal, April 14, 1900.
3. Discourse delivered by Dr. Ronald Ross before the Royal Institution of Great Britain, March 2, 1900.
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5. Marchiafava and Bignami, in Twentieth Century Practice of Medicine, 1900, vol. xix.
6. Notes on the Mosquitoes of the United States, by L. O. Howard, U. S. Dept. of Agriculture, Bulletin 25, New Series, 1900.
7. Report of the Malaria Expedition to Africa, made by the Liverpool School of Tropical Medicine (Ross, Annett, Giles, and Fielding-Ould), 1900.
8. Presented before the Philadelphia Pathological Society, November 15, 1900.

DISCUSSION.

DR. KEEN: This is the second observation in which the malarial parasite had been cultivated in America. The subject is of special interest to the Fellows of the College.

A vote of thanks was tendered to Dr. Woldert for the report of his valuable investigations.

DESCRIPTION OF PLATE.

Four zygotes (unstained) of æstivo-autumnal malarial parasites in different stages of development, and found in lower portion of middle intestine (stomach) of Anopheles quadrimaculata (or claviger). The mosquito bit on Thursday, November 1st, 8 to 9 P.M., and was dissected on the Monday following. No. 3 oc., No. $\frac{1}{12}$ oil-immersion lens (Leitz).

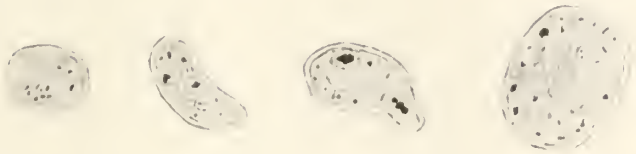
No. 1. Ovoid-shaped zygote in an early stage of development, and containing numerous reddish pigment granules and vacuoles. About 14 to 16μ in diameter.

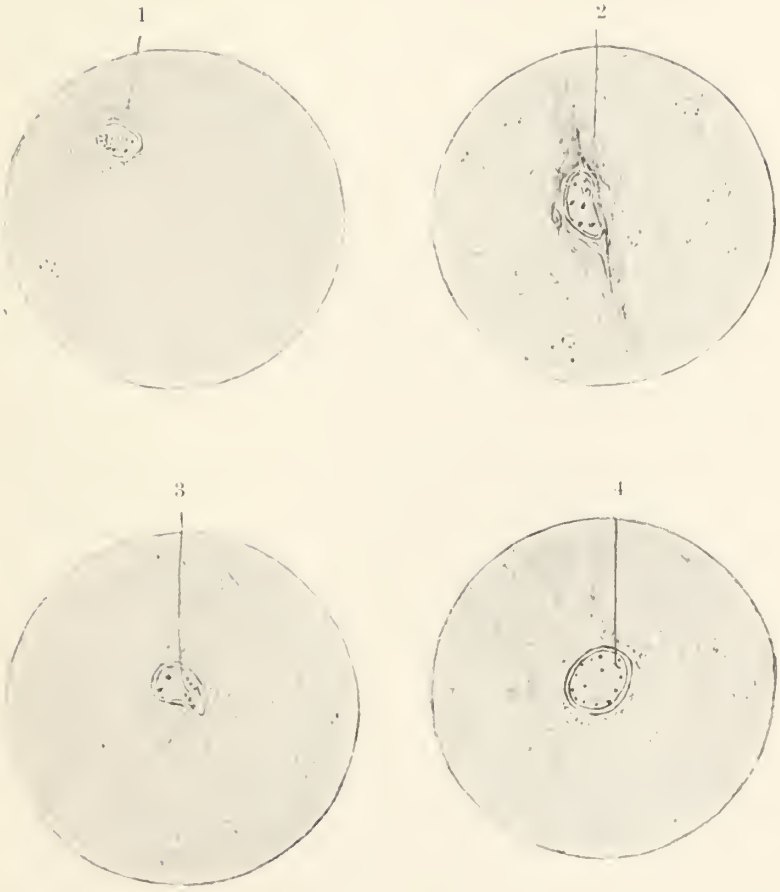
No. 2. Crescent-shaped zygote containing reddish pigment granules and growing between the muscular fibres, which it pushes to either side. The pigment granules are of larger size than in No. 1, and are pressed toward the periphery. About 14 to 16μ in length.

No. 3. More or less vesicular-shaped coccidium containing bright reddish pigment granules and slit-like vacuoles. The body of the coccidium has a faint yellowish tint. It is about 10μ in diameter and 14 to 17μ in length.

No. 4. More or less ovoid-shaped zygote containing numerous reddish pigment granules of destroyed hæmoglobin which are arranged around the periphery. Numerous slit-like vacuoles are also shown. It is about 12 to 14μ in diameter and 16 to 20μ in length.

Figs. 1, 2, 3, and 4 greatly enlarged.

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APPENDIX.

ADDITIONS TO THE MÜTTER MUSEUM, 1900.

Aneurism of the Aorta. Presented by Dr. Oscar H. Allis.

Fracture of the Skull. Presented by Dr. Oscar H. Allis.

Resection of the Elbow-joint. Presented by Dr. Oscar H. Allis.

Necrosis of the Bones of the Foot and Leg. Presented by Dr. Oscar H. Allis.

Old Luxation of the Hip-joint. Presented by Dr. Oscar H. Allis.

Skull showing Absorption of both Orbits. Presented by Dr. Oscar H. Allis.

Inferior Maxilla of a Sheep. Presented by Dr. Oscar H. Allis.

Cast of a Fracture of the Wrist-joint. Presented by Dr. Oscar H. Allis.

Splint for Excision of the Knee-joint. Presented by Dr. De Forest Willard.

Splints for Fracture of the Wrist. Presented by Dr. De Forest Willard.

Twenty-four Skiagraphs. Presented by Dr. De Forest Willard.

A Collection of Calculi. Presented by Dr. De Forest Willard.

Sixty Surgical Specimens. Presented by Dr. De Forest Willard

A Fœtus of Three Months. Presented by Dr. Charles Baum.

Painting of Heloderma Suspectum. Presented by Dr. S. Weir Mitchell.

Dr. Mütter's Gold-headed Cane. Presented by Dr. S. Weir Mitchell.

Four Sections of Melanosarcoma of the Choroid. Presented by Dr. Charles A. Oliver.

A Collection of Laryngological Specimens. Presented by Dr. J. Solis-Cohen.

Triple Fœtuses. Presented by Dr. A. P. Charlton.

Two Photographs of a Monster. Presented by Dr. J. V. Ingham.

Photograph of a Double Baby. Presented by Dr. J. V. Ingham.

Thirty-six Vesical Calculi. Presented by Dr. John Ashhurst, Jr.

Aortic Aneurism. Presented by Dr. F. A. Packard.

Hypertrophied and Dilated Heart. Presented by Dr. F. A. Packard.

Specimens from a Case of Aortic Obstruction. Presented by Dr. F. A. Packard.

A Collection of Pathological Specimens. Presented by Dr. F. A. Packard.

Corrosion Preparations of the Liver and Lungs. Presented by Dr. F. M. Paul.

Human Fœtus. Presented by Dr. F. M. Paul.

Three Wooden Stethoscopes. Presented by Dr. Geo. I. McKelway.

Femoral Tourniquet. Presented by Dr. Geo. I. McKelway.

Death Masque of John Hunter. Presented by Dr. W. W. Keen.

Gavel Presented to Dr. Keen as President of the American Medical Association. Presented by Dr. W. W. Keen.

A Large Collection of Surgical Instruments. Presented by Dr. J. Ewing Mears.

Twenty Replicas of Surgical Instruments from the Ruins of Pompeii. Presented by Dr. Joseph Leidy.

ABSTRACT OF THE REPORT OF THE LIBRARY COMMITTEE FOR 1900.

The Library contains 61,359 volumes—an increase of 4158—2611 greater than the increase reported last year.

There are 4140 duplicates not yet disposed of, which brings the total number of volumes to 65,499. There are also 46,530 unbound pamphlets, reports, and transactions.

The Library receives regularly 301 periodicals—85 American and 216 foreign. Thirty journals have been added to the subscription list. Three American and six foreign publications have been added to the exchange list, and arrangements for exchange of publications have been made with the Universities of Berlin, Strasburg, Bonn, and Halle, and the Faculties of Medicine of Nancy and Rio de Janeiro.

Six thousand nine hundred and sixty-three inaugural dissertations have been received.

Thirty-three of the new publications added to the Library have been written or edited by Fellows of the College.

There have been 4781 visitors to the Library. Ten thousand four hundred and forty-two books have been furnished by the Librarian for consultation, in addition to those taken from the shelves by Fellows. Two housand four hundred and eleven books have been taken out.

George C. Harlan,
Chairman.

LIST OF PAPERS: SECTION ON OPHTHALMOLOGY.

December 19, 1899.

Case of Traumatic Varix of the Orbit in which Ligation of the Left Common Carotid Artery was Successfully Performed, by Dr. C. A Oliver.

Traumatic Rupture of the Choroid, with Hemorrhage from the Upper Branch of the Inferior Temporal Vein, with Water-color Sketches, by Dr. C. A. Oliver.

Prolapse of the Iris after Simple Cataract Extraction, by Dr. G. C. Harlan.

January 16, 1900.

Exhibition of a Card Showing Compact Arrangement of Jaeger Test-types, by Dr. S. Lewis Ziegler.

Persistent Pupillary Membrane, with Exhibition of Case, by Dr. A. G. Thomson.

Case of Blindness from Sympathetic Ophthalmitis, with Restoration of Vision by Critchett's Operation, by Dr. G. E. de Schweinitz.

A Series of Complicated Cataracts, by Dr. G. E. de Schweinitz.

Oculomotor Paresis following Indirect Violence, by Dr. C. A. Veasey.

A Case of Transient Real Blindness, by Dr. G. C. Harlan.

Double Optic Atrophy from Otitic Thrombosis, by Dr. B. A. Randall.

February 20, 1900.

Mental Disturbances after Operations upon the Eye, by Dr. W. C. Posey

Concerning the Preparation of the Stump after Complete Enucleation of the Eyeball, by Dr. G. E. de Schweinitz.

A Study of Eighteen Cases of Foreign Body in the Eyeball, by Dr. Charles Lukens (by invitation).

A Foreign Body (Metal) which Remained Quiescent in the Choroid of a Practically Blind Eye for Eighteen Years, by Dr. G. E. de Schweinitz.

A Study of the Changes in Refraction in Four Hundred Eyes during Seven Years, by Dr. H. F. Hansell.

A Case of Diffuse Punctate Condition of the Fundus, by Dr. E. A. Shumway (by invitation).

March 20, 1900.

Exhibition of a Case of Bilateral Enlargement of the Lacrymal Glands, by Dr. W. C. Posey.

Exhibition of a Case of Synchysis Scintillans, by Dr. A. G. Thomson.

Traumatism from the Lash of a Whip, with Exhibition of Case, by Dr. C. A. Oliver

Iridectomy with Removal of Lens Capsule and Lens Débris in a Case of Blindness of more than Thirteen Years, by Dr. C. A. Oliver.

The Relation of Tenon's Capsule and the Check Ligaments to Enophthalmos, by Dr. William T. Shoemaker.

Concerning the Filling of Collapsed Eyeballs with Physiological Salt Solution, by Dr. G. E. de Schweinitz.

A Case of Left Hemianopsia, with Alexia, by Dr. S. D. Risley.

History of a Case of Partial Paralysis of the Third Nerve, by Dr. S. D. Risley.

April 17, 1900.

Exhibition of a Cast of Solder Removed from the Conjunctival Sac in a Case of Traumatism, by Dr. H. F. Hansell.

Sympathetic Inflammation Occurring more than Two Months after Enucleation, by Dr. W. Zentmayer.

Glaucoma in Myopia, by Dr. John T. Carpenter.

Complete Restoration of the Conjunctival Sac by a Single Skin Graft, by Dr. C. A. Oliver.

A Clinical and Pathological Report of Three Cases of Secondary Glaucoma, by Drs. W. C. Posey and E. A. Shumway.

A Clinical and Histological Study of a Case of Melanotic Sarcoma of the Choroid, by Dr. C. A. Oliver.

Monocular Hysterical Amaurosis, by Dr. C. A. Veasey.

A Cured Retinal Detachment, with Remarks on Retinitis Striata, by Dr. G. E. de Schweinitz.

October 16, 1900.

Removal of Metallic Foreign Bodies, with Exhibition of Two Cases, by Dr. William Thomson.

Embolism of the Central Retinal Artery, with Exhibition of Case, by Dr. G. C. Harlan.

Simple Extraction of Cataract, with Exhibition of Case, by Dr. S. D. Risley.

A Piece of Glass in the Ciliary Body Located by the Röntgen Rays and its Removal with Forceps, by Dr. William M. Sweet.

Concerning Traumatic Palsies of the Ocular Muscles, with Cases, by Dr. G. E. de Schweinitz.

November 20, 1900.

Abscess of the Orbit following Injury, with Exhibition of Case, by Dr. S. D. Risley.

Papilloma of the Caruncle, by Drs. W. C. Posey and E. A. Shumway.

Two Cases of Intraocular Metallic Foreign Bodies, by Dr. G. E. de Schweinitz.

A Word Concerning the Etiological Relationship of Epidemic Influenza to Chronic Glaucoma, by Dr. G. E. de Schweinitz.

Report of a Case of Left Lateral Homonymous Hemianopsia, by Dr. M. W. Zimmerman.

A Case of Traumatic Luxation of the Crystalline Lens; Secondary Glaucoma; Extraction without Loss of Vitreous; Recovery with Normal Vision, by Dr. C. A. Veasey.

A Case of Hemorrhage from the Conjunctiva in an Infant, by Dr. H. F. Hansell.

WILLIAM M. SWEET,
Clerk of Section.

LIST OF PAPERS: SECTION ON OTOLOGY AND LARYNGOLOGY.

Exhibition of a Case of Nævus of the Tympanum, by Dr. E. B. Gleason.

An Unusual Case of Papilloma of the Larynx, with patient; and also a Case of Laryngeal Growths for Diagnosis, by Dr. Joseph S. Gibb.

Treatment of a Common Cold, by Dr. Frank Woodbury.

Osteophytes of the Nasal Chambers, by Dr. Alexander W. MacCoy.

The Silver Salts in the Treatment of Chronic Suppuration of the Middle Ear, by Dr. Edward B. Gleason.

A Few Suggestions in the Treatment of Atrophic Rhinitis, by Dr. Arthur W. Watson.

A Case of Atrophic Rhinitis Associated with the Presence of Bacillus Subtilis, by Dr. Frank Woodbury.

A Case of Nasopharyngeal Mycosis, with patient, by Dr. P. S. Donnellan.

Three Recent Cases of Mastoid Abscess Breaking into the Digastric Fossa (Bezold Cases), One a Child, by Dr. B. Alexander Randall.

Report on Cases of Foreign Bodies in the Nasal Chambers, by Dr. W. R. Hoch.

Grave Diseases in Young Children Often Due to Latent Inflammation of the Middle Ear, by Dr. Charles H. Burnett.

Hot-air by Electricity, by Dr. D. Braden Kyle.

Syphilitic Disease of the Auricle, by Dr. Frederick A. Packard.

Mycosis of the Faucial and Lingual Tonsils, with Case, by Dr. W. R. Hoch.

A Case of Laryngeal Carcinoma and One of Healed Tubercular Ulceration of the Larynx, by Dr. P. S. Donnellan.

A Case of Acute Otitic Paralysis, by Dr. Charles H. Burnett.

Report of Operation Within the Nose and Nasopharynx in a Case of Hæmophilia, by Dr. C. P. Bliss.

Report of a Case of Mastoiditis, with Unusual Clinical History, by Dr. Ralph W. Seiss.

A Case of Facial Paralysis Occurring in Acute Otitis Media Tuberculosa, by Dr. Charles H. Burnett.

On Chancre of the Tonsil, by Dr. George C. Stout.

Myxoma of the Larynx, by Dr. Joseph S. Gibb.

Presentation of Cases: (1) Mastoid Abscess with Extra-dural Abscess; (2) Purulent Thrombosis of the Lateral Sinus, by Dr. B. Alexander Randall.

Presentation of a Patient with Hernia Cerebelli following an Operation for Mastoid Disease, by Dr. B. Alexander Randall.

FRANK WOODBURY,
Clerk of Section.

LIST OF PAPERS: SECTION ON GENERAL MEDICINE.

DECEMBER, 1899, TO DECEMBER, 1900.

Cases Shown.

Case of Splenic Anæmia, by Dr. Alfred Stengel.

A Case of Myxœdema which has not been benefited by Thyroid Treatment, by Dr. M. Howard Fussell.

A Case of Hysterical Monoplegia Cured by Suggestion, by Dr. F. Savary Pearce.

(1) A Case of Unilateral Progressive Ascending Paralysis, with Exaggerated Scapulohumeral Reflex (v. Bechterew); (2) A Case of Pseudorhizomelic Spondylosis, by Dr. William G. Spiller.

A Case of Myxœdema, with Complications, by Dr. M. H. Fussell.

Specimens Exhibited.

A Case of Mitral Stenosis, with the Exhibition of a Specimen, by Dr. J. A. Scott.

Some Intestinal Casts, by Dr. F. A. Packard.

Papers Read.

The Position and Color of the Ring in Heller's Test for Albumin in the Urine, by Dr. M. Howard Fussell.

Withdrawal of Liquids from the Diet of Diabetes Mellitus, by Dr. Joseph Sailer.

An Ovarian Cyst Associated with Diabetes; Operation followed by Disappearance of the Diabetes, by Dr. H. D. Beyea.

Friedreich's Ataxia, with Report of a Fatal Case, by Dr. F. Savary Pearce and Dr. J. M. Swan.

A Case of Rheumatic Pericarditis with Serofibrinous Exudate; Recovery, by Dr. Herman B. Allyn.

A Case of Aortic Aneurism in which Electrolysis was Performed Three Times, by Dr. Hobart A. Hare.

The Definition and Significance of Accentuation of the Second Pulmonary Sound, by Dr. Alfred Stengel.

Diet in Typhoid Fever, by Dr. F. A. Packard.

The Use of Stimulants in Typhoid Fever, by Dr. J. H. Musser.

The Use of Intestinal Antiseptics in Typhoid Fever, by Dr. J. M. Anders.

The Importance of Glycosuria as a Symptom, by Dr. Alfred Stengel.

The Relations of Diet to Diabetes, by Dr. David L. Edsall.

The Treatment of Diabetes and its Complications, by Dr. James Tyson.

A Case of Compression of the Upper Part of the Cervical Cord, with Unusual Symptoms, by Dr. William G. Spiller.

The Types of Anæsthesia Occurring in Pott's Disease, by Dr. C. W. Burr.

Angina Pectoris, by Dr. A. A. Eshner.

Dislocations of the Heart in Cases of Pleural Effusion, by Dr. M. H. Fussell.

An Unusual Influence of Strychnine in Diseases of the Kidneys, by Dr. Walter I. Pennock.

Splenic Anæmia, by Dr. A. O. J. Kelly.

Two Cases in which the Signs of Mitral Stenosis Disappeared as Cardiac Compensation was Re-established, by Dr. M. H. Fussell and Dr. J. D. Steele.

Diabetes Mellitus as a Cellular Fault, by Dr. Thomas C. Ely.

Abscess of the Liver; Hepatotomy; Recovery, by Dr. David Riesman.

A Physician's Holiday at Carlsbad, by Dr. James Tyson.

SAMUEL McC. HAMILL,
Clerk of Section.

LIST OF PAPERS: SECTION ON GYNECOLOGY.

January 18, 1900.

A Case of Nephrectomy for Stone, by Dr. Charles P. Noble.

The Pernicious Nausea and Vomiting of Pregnancy, with the Report of a Case, by Dr. Edward P. Davis.

February 15, 1900.

Dermoid Cystoma of the Pelvic Connective Tissue, with Report of a Case, by Dr. H. D. Beyea.

Report of Two Cases of Extra-uterine Pregnancy:

(*a*) Tubo-ovarian Pregnancy, following Relapsing Appendicitis.

(*b*) Ruptured Tubal Pregnancy, with Symptoms of Infection, Treated by Vaginal Puncture, by Dr. John G. Clark.

Remarks upon the Operative Treatment of Uterine Prolapse, by Dr. John B. Deaver.

Exhibition of Specimens:

Epithelioma of the Cervix Uteri, Removed by Vaginal Hysterectomy, by Dr. John B. Shober.

March 15, 1900.

Some Recent Experiences in Intestinal Surgery, by Dr. B. C. Hirst.

A Multilocular Ovarian Cyst, with Peculiar Course, by Dr. G. G. Davis.

April 19, 1900.

A Report of Two Cases of Epithelioma of the Vulva, by Dr. Charles P. Noble.

A New Intestinal Anastomosis Forceps, by Dr. M. O'Hara, Jr.

(1) Ovarian Cyst (32 Pounds) Removed Six Weeks after a Normal Labor.

(2) Traumatic Rupture of a Dermoid Cyst, by Dr. Robert G. Le Conte.

Special Meeting, April 30, 1900.

Periods in Gynecology, by Dr. H. J. Garrigues, of New York (by invitation).

May 17, 1900.

A Case of Secondary Papillary Cyst-adenoma of the Posterior Wall of the Vagina, by Dr. H. D. Beyea.

The Rôle of the Liver in the Production of Eclampsia, by Dr. W. A. N. Dorland.

October 18, 1900.

A Case of Ruptured Tubal Pregnancy, by Dr. E. P. Davis.

A Case of Extra-uterine Pregnancy, by Dr. H. D. Beyea,

November 15, 1900.

(1) Unattached Body in the Abdominal Cavity, Probably Ovary.

(2) Combined Carcinoma and Fibroma of the Uterus, by Dr. George Erety Shoemaker.

(1) Atresia of the Uterus.

(2) Cæsarean Section for Carcinoma of the Rectum, by Dr. J. M. Baldy.

JOHN H. GIRVIN,
Clerk of Section.

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